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ADVANCES IN SURGERY DURING THE PAST 40 YEARS*

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Fellow members of the Surgical Association of Louisiana, guests, ladies, and gentlemen: First of all, I want to thank you for having bestowed upon me the highest honor of this Association. It is a great pleasure to be here in New Orleans, which City I have always regarded as my second home, and to address you on this august occasion.

I see amongst you some of my oldest and dearest friends whom I love to see from year to year. However, my joy is tempered by a feeling of sadness when I no longer see some of the old familiar faces who have gone to that land from whence no traveler returns. However, this is life. Like the flowers, they bud, bloom, and fade away.

When one has a choice of subject on which to speak, it is difficult to make a decision.

I was reminded of the story of a tramp who applied to a farmer for work. It so happened that the farmer was in need of help and a bargain was made. The farmer said, "Now the first job I want you to do is cut and cord this pile of wood. I will be back around noon and see what you have done." On his return he found that the tramp had finished the job in record time. "Now that is fine—your job for tomorrow is to sort this big pile of potatoes into three piles putting the small ones in one pile, the medium sized in a second and the large ones in a third pile. I will be back at noon to see what you have done." On his return he was disappointed to find that almost nothing had been done and the tramp was sitting down with his head in his hands. The farmer said, "I can't understand why you did such a good job yesterday with the wood and today such a poor job sorting the potatoes." The tramp replied, "Well, the work is all right, but making these decisions is killing me."

I decided to reminisce on the progress I have observed in surgery during the past forty years. In passing, I should like to briefly make a few remarks on the history of medicine.

"Man has climbed up from some lower animal form," says John Burroughs, "but he has, as it were, pulled the ladder up after him."

Our knowledge of ancient medicine is practically nil, but it is highly probable that in all primitive societies, the priest, the magician, and the medicine man were one and the same. As these functions became more specialized and differentiated, religion became the exclusive belief in, and the worship of some universal power greater than man himself.

Magic was a special set of processes within the power of man whereby he sought to predict and control natural phenomena, usually to wreak evil and to work in opposition to the will of the god or gods, and medicine was the attempt to direct and control those natural phenomena which produce disease and death in man.

This religion, through the inhibitions which man put upon himself to attain the

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godlike, became the origin of law and ethics; the secret practices of magic engendered alchemy and other branches of chemical and physical sciences, astrology, and astronomy; while primitive medicine remained more or less stationary among all peoples, always following in the wake of other sciences, until it could utilize the advances made by physics or chemistry.

Garrison, in his history of medicine, quotes Herodotus on the practice of medicine among the Babylonians as follows:

"They bring out their sick to the market place for they have no physicians; then those who pass by the sick person confer with him about the disease to discover whether they have themselves been afflicted with the same disease as the sick person, or have seen others so afflicted, thus the passers-by confer with him, and advise him to have recourse to the same treatment as that by which they escaped a similar disease, or as they have known to cure others. And they are not allowed to pass by a sick person in silence, without inquiring into the nature of his distemper."

I might add that in spite of the great advances in medicine and the array of specialists, the Babylonian system has been handed down to the present day. Well meaning friends and neighbors are eager to diagnose and advise treatment of any malady.

Evidence unearthed by archeologists would indicate that surgery was in advance of medicine. While medicine consisted largely of mineral and vegetable simples compounded with such filthy ingredients as the blood, excreta, fats and visceral parts of birds, mammals and reptiles, surgery was done with the neolithic chipped knife. Circumcision and craniotomy were probably among the earliest operations and apparently the ancients set and splinted fractures with a remarkable degree of skill when you consider that they were dependent entirely on seeing and feeling.

Some ethical precepts of the ancient Egyptian physicians are very much like the Hippocratic Oath in sentiment and expression, and this alone would point to the fact that pre-Hippocratic medicine in Greece had an origin closely connected with Egyptian medicine.

The real foundation and ethics of modern medicine had its beginning with Hippocrates, a Greek, about three or four hundred years before Christ and he is referred to as the "Father of medicine and the greatest of all physicians." Although surgery made great strides, its usefulness was limited by lack of antisepsis and anesthesia.

Koch, Pasteur and Lister laid the foundation of antisepsis in the middle of the 19th century and although it was crude, surgeons became bolder and began to invade the visceral cavities. Theodore Bilroth is credited with being the pioneer of visceral surgery, about 1860.

Time will not permit me to relate more of the ancient history of medicine so I shall now start on the forty year period with which I am familiar. When I entered Charity Hospital in New Orleans, in 1914, as an intern, rubber gloves were not too plentiful and were used in the operating rooms only. Gynecologic examinations rooms only. Gynecologic examinations were done barehanded and I recall that one of my fellow interns developed a primary lesion of syphilis on the index finger of the right hand. Treatment of syphilis was limited to mercury and iodides. This was painful and the duration of treatment was three years. In this connection I remember an expression of my good old professor, Dr. Metz, "Three minutes at the shrine of Venus and three years at the shrine of Mercury."

In the early part of 1915, we first used salvarsan better known as 606. This was quite a ritual as it had to be properly mixed in the hospital drug store and given in about a liter of water. A cut down on the vein and canula was routinely used. Why some one had not thought of venepuncture, as we now give intravenous fluids and medication, is hard to understand.

For postoperative patients who were unable to retain fluid given orally, we used proctoclysis. It was called the Murphy

drip, but it is my belief that our own beloved Professor Rudolph Matas used it before Murphy. Dr. Matas prescribed the drip using black coffee or Coca Cola and glucose in tap water. This was our only means of hydrating patients and while it did save some lives it was a messy affair. Usually the patient was or became distended and the drip was returned to the bed with compound interest. It was quite some years later before a safe and practical means was evolved to give fluid and electrolytes intravenously. At the present time there is but one good use for the Murphy or Matas drip and that is for fecal impaction when the return with the aforementioned compound interest is gratefully received by the patient and the doctor.

For anesthesia, chloroform was rarely used except in Obstetrics, and the ACE mixture (alcohol, chloroform and ether) was on the way out in favor of straight drip ether.

There were no professional anesthetists and all anesthetics were given by interns or members of the surgical staff dripping ether for each other.

Spinal anesthesia was used to a limited degree, but was not in great favor. The drug used was one grain of tropococain. Usually the patient had a chill and fever 104 to 105° with severe headaches. Many left the hospital taking with them the headache and it was not rare for one to leave for parts unknown. Any resemblance of spinal in those days to spinal nowadays with the improved drugs and expert anesthetists is purely coincidental.

Local anesthesia was used very little, as cocaine was highly toxic and had to be used very sparingly. Although procaine was first made by Alfred Einhorn in Germany in 1904, it did not come into general clinical use until a number of years later. Our own Dr. Carrol W. Allen was one of the pioneers in this field.

Chest surgery was practically limited to thoracotomy for empyema. Empyema was very common and we did many thoracotomies before the advent of the antibiotics.

Blood transfusions had to be given by the direct method, having donor and recipient side by side in the operating room. It was a tedious job using a Joubet transfuser. Blood would clot in the recipient or donor needle or both. After a few syringefuls the plunger would stick in the barrel requiring cleansing. While this was being done, blood would clot in the needles and necessitate starting from scratch. Fresh needles had to be introduced into the veins of both donor and recepient. You were better than average if you could transfuse half a pint and expert if you could manage one pint.

The citrate method and blood banks emancipated transfusion making it possible to give large quantities of whole blood and perform operations consuming four to eight hours time. Before this we considered operations hazardous which extended beyond two hours.

The great improvement in anesthesia by a closed system with inlying intratracheal tube has made it possible to do chest surgery which was considered almost certain death twelve years ago.

The discovery of insulin by Banting and Best, in 1921, has made it possible to prepare and operate on diabetics who would most certainly have died previously.

Until the advent of the antibiotics, only a few years ago, osteomyelitis was a serious and disabling disease requiring extensive surgery which frequently was fruitless and futile.

I recall during my intern days at Charity Hospital in the right wing of the second floor of the main building, we had numerous pus cases, notably compound fractures, which we irrigated with bichloride of mercury solutions. This went on day in and day out with very disappointing results. This area was referred to as "Pus Alley," a name which it richly deserved. During my time on this service, I had two colored boys—one with compound fracture of the right arm and the other with compound fracture of the left arm. Both drained pus profusely and both boys looked very much alike. I named

them Jubilee and Jubilum. A few years later, after I began practicing medicine in Baton Rouge, a one armed Negro boy came to my office. He said, "Doctor does you remember me?" I didn't and he said, "I am Jubilee." I then remembered him and inquired about Jubilum. He said Jubilum died. Well that was just about our mortality and morbidity in those days.

I have been privileged to practice in an era that has witnessed many changes and improvements in anesthesia and antisepsis, however, there has not been much change in surgical technique as we have had masters of the art during and before my time.

I recall about 1913 or 1914, I was sitting in the Miles Amphitheater watching our own Dr. Urban Maes, then a very young visiting surgeon, perform an operation. Sitting next to me was a man in shirt sleeves and wearing red suspenders. He remarked, "That young man has a beautiful surgical technique." I later learned that the gentleman was none other than Dr. Charles Mayo.

Fractures of the hip were treated by the Whitman method. That is by abduction, interal rotation, and a plaster cast extending from the nipple line to the toes on the injured side and to just above the knee on the well side. Many of these patients died while incarcerated in the cast and those who survived had stiff joints that required months of physical therapy to restore motion. The alternative to the Whitman procedure was to place sand bags on either side of the injured leg and give morphine as required for pain. The patients developed bed sores, were miserable, and were happy to be escorted away by what Sir William Osler called the old folks' friend—pneumonia.

In New Orleans, we had pioneers in the field. Dr. Denegre Martin had successfully used large carpenter's screws for fixation of the hip and Dr. Parham used his band on certain fractures of the long bones.

Our own Dr. Isadore Cohn appreciated the work of Martin and continued to use the large carpenter's screws with the added refinements of stainless steel and x-ray guidance. As a matter of fact, his work was so successful that he never, to my knowledge, switched to Smith-Peterson or any of the other more recent instruments of fixation.

In 1935, I spent a short time in Chicago observing Drs. Cubbins and Callaghan doing hip nailing. I then observed Dr. Willis Campbell in Memphis, after which I felt confident that I could nail hips. I bought the necessary equipment and returned home.

A few days later an elderly lady was admitted to the hospital with an intracapsular fracture of the hip. I had a hard job selling her family on the operation, but succeeded two or three days later. Although I had no fracture table and only a 10 milliampere portable x-ray machine using a 110 volt current which sometimes dropped to 80 or 90 volts, I had the good fortune of doing a good job.

About a week later, I had a second case but could not sell the family on the idea of an operation. My first case was doing well and in a rolling chair, so I had her nurse roll her in to talk to the second case. The family was amazed to find a patient in a rolling chair with no cast, who had broken her hip only a week ago, and readily consented to operation. My good fortune continued and I was doing a thriving hip business until one day a very elderly man was admitted with an intertrochanteric fracture and acutely ill with pneumonia. I had to do him under local anesthesia and I had no suitable trochanteric extension. He was delirious and it was difficult to restrain him on his return to his room. Needless to say, my work was put asunder. I then resorted to well leg traction which held the fragments in good apposition. The old man recovered, but I was never able to get union and he lived to bear witness to my failure. I received plenty of adverse advertisement. I was thankful that he had not been my first case and I was able to live through this, my first bad result. At that

time Baton Rouge was like the Dutchman's pigs, little, but old. We had no specialist except in eye, ear, nose and throat and pediatrics. At the present time, we are a thriving city of some 135,000 population with all the special fields covered by well trained men.

In my early days we had no Fellowships, but had to learn by preceptorship and experience. One started as general practitioner and worked into his chosen field. The one good feature was that it did give a broad perspective of the patient as a whole.

At the present time, medicine has become so broadened in its scope that no one brain in a normal lifetime can experience and retain the experience of every field of our art. However, the vast majority of conditions can be handled very well and much more economically by good general practitioners who can sort out and refer the unusual things to the proper specialists. The general practitioner has to be well trained and to cover so much territory with very little of the glamor and with smaller fees, but he is indispensable. He may be likened to the private in the army without whom no war could be fought.

The young men of today are fortunate in that so many provisions have been made for special training of which the public is familiar. He is no longer viewed with suspicion and held in abeyance until his hair has grayed to serve as a mark of experience.

I have often heard the expression used "living in the good old days." Well I have lived long enough to experience both eras and I can say with one exception, income tax, we are now living in the good days. I am happy to have lived to enjoy the great advances in medicine, to ride in an automobile, to draw good water from a faucet, to have light by snapping a switch, to have the comforts of heating and cooling, refrigeration of food, and things too numerous to mention.

Last but not least, I enjoy the companionship of the well trained young men with whom I associate and from whom I have learned much. I do hope that in turn, I have been of some help to them.

To recount the advances surgery has made reminds me of the colored pastor who chose to talk to his congregation on the advancement of speed in the past fifty years. He began by saying that in 1900 a good horse hitched to a buggy carried you about seven miles per hour. One of the faithful sitting on the front row said "Amen." Next he said came the automobile making about fifteen or twenty miles per hour. Another "Amen" from the faithful. Later with good roads and improved cars, we made fifty to seventy miles per hour—"Amen." Then came the aeroplane making one hundred to one hundred fifty miles per hour. "Amen." Now we got the jet planes making seven or eight hundred miles per hour—"Gawd Dam!"

THE MANAGEMENT OF URETERAL CALCULI*

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It is not my purpose to review the literature or to bore you with statistics dealing with a series of cases in effort to discuss the problem of ureteral lithiasis. The former is common knowledge to all of you, but the absence of the latter may indeed make this discussion seem a bit philosophical. In spite of this, however, I am simply going to discuss certain ideas which I have with respect to ureteral lithiasis and its management.

DIAGNOSIS

Since most ureteral stones have their origin in the calices or renal pelvis and then course down the ureter, the characteristic story of costovertebral pain with somatic shifting of its location, as it progresses down the ureter, is classically obtained. A carefully taken history is the single most important factor in establishing the diagnosis of ureteral lithiasis. On

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the other hand, the patient may relate no such classical story but may present complaints commonly associated with trigonal irritation, suggesting cysto-urethritis in the female or vesical neck obstruction in the male. On occasion, I have seen patients subjected to prostatic surgery, protracted courses of prostatic massage, or fulguration of the urethrovesical junction, when in reality the agent responsible for the symptoms was that of a calculus located at the intramural ureter. In fact, symptoms pointing to trigonal irritation may present initially, without the appearance of colic, when a small stone has passed down a relatively large ureter producing no obstruction in transit. Gradual ureterectasis with accompanying renal damage may then take place resulting in progressive hydronephrosis and rather insensible renal destruction.

The relative age of the patient is of some assistance in diagnosis of a ureteral calculus in that calculi are more commonly seen in early adulthood. Gross hematuria, though by no means constant, may present. Systemic manifestations of chills and fever depend, to a large measure, on the amount of infection proximal to the point of obstruction.

PHYSICAL FINDINGS

The findings on physical examination vary from exquisite loin tenderness, as seen in acute severe ureteral obstruction, to a complete absence of any findings whatsoever. A marked degree of obstruction is frequently accompanied by a moderately elevated blood pressure.

Depending upon the amount of irritation and infection present, together with the completeness of obstruction, pyuria and a varying amount of microscopic hematuria may be noted. These factors also influence the leukocyte count.

X-RAYS

Of paramount importance, when suspecting a ureteral stone, is the plain film of the abdomen, taken so as to include the kidneys, ureters and bladder made in conjunction with an excretion urogram. The plain film may or may not present a shadow in line with the ureter. The absence of such a shadow by no means excludes the possibility of ureteral lithiasis. The excretion urogram is second in importance to the history in establishing a diagnosis. It allows one to determine the degree of obstruction present, the exclusion or inclusion of certain shadows along the course of the ureter, and often enough, assists in accentuating the shadow cast by a calculus of low radiopacity provided ureteral obstruction is not complete. An angulated film should always be made at the time of adequate ureteral filling in order to accurately include or exclude shadows in question along the course of the ureter.

Retrograde pyelography, in the absence of preliminary excretion urography, is mentioned only to be roundly *condemned*. Furthermore, I have never felt that it should be employed to establish a diagnosis of ureteral lithiasis. If a pyeloureterogram is to be made it should be deferred until several days after removal of the calculus and after institution of ureteral drainage. The forcing of pyelographic media by a point of obstruction is simply to court disaster. Cortical abscesses of the kidney associated with fulminating pyelonephritis and septicemia do not pose the problem they once did, owing to our present antibiotic and chemotherapeutic armamentarium. Yet, I see no need for inviting trouble.

Often one must rely on the history alone to establish a diagnosis of ureteral lithiasis. Frequently it has been the experience of all of us to manipulate and remove a ureteral calculus in the absence of positive findings on physical examination, urinalysis, or as evidenced on roentgenographic study.

MANAGEMENT

The excretion urogram is of inestimable value in determining the course of management to be pursued. If the degree of obstruction is minimal or completely absent one may temporize. If, on the other hand, the amount of obstruction is marked to complete, relief from the obstruction either by passage of ureteral catheters by

the point of obstruction with fixation of the same in place to initiate drainage or actual removal of the calculus becomes mandatory.

The degree of obstruction present, the size and location of the calculus, and finally, the desire of the patient determine the approach to the problem.

As mentioned, in the presence of a high degree of obstruction, it is necessary to decompress the obstructed kidney if renal damage is to be prevented. This may be done either by removal of the stone or by tying in ureteral catheters which have been passed beyond the point of obstruction. The latter course is occasionally necessary in effort to better prepare the debilitated patient, prior to undertaking more definitive action. It is well to explain to the patient, prior to cystoscopy, that the calculus may be forced back into the renal pelvis at the time of catheterization or manipulation, a result which if not well understood, may aggravae the entire management of the problem.

In general, calculi confined to the upper half of the ureter, requiring prompt attention, are removed through a small muscle-splitting incision placed so as to come down directly on the point of ureteral obstruction. On the way to surgery a plain film of the abdomen is repeated in order to make certain of the exact position of the calculus at the time of exploration. The Lloyd-Lewis forcep has proven very useful in fixing the stone in the ureter, assisting in its delivery when the ureter is cut down upon. The ureter is rarely closed. With the muscle-splitting procedure convalescence is reduced to a bare minimum complicated only by rather free drainage of urine from the operative site until the incised ureter has had opportunity to seal over, usually requiring four to five days.

Calculi located in the lower half of the ureter, and more especially in the lower third, are with a rare exception removed through manipulative effort. While there are numerous instruments and devices which have been developed, the Johnson and Councill extractors have proven to be most useful in my hands. The looped catheter has been used with good result in manipulation of ureteral calculi in children. Occasionally one encounters a calculus, of large dimension, considered too large to be manipulated, requiring an open surgical effort. Intramural ureterotomy is ordinarily unnecessary in order to deliver the calculus into the bladder.

It is at times wise to do an initial cystoscopic examination, tying in ureteral catheters solely to produce dilatation about the calculus before undertaking manipulation. Several days later, at a second sitting, attempt is made to manipulate and remove the calculus. Formerly this was the rule rather than the exception. More recently, possibly owing to the added sense of security offered by newer chemotherapeutic and antibiotic agents, the majority of calculi have been manipulated initially. After calculus removal, two number five catheters are tied in the ureter, together with a Foley bag catheter in the bladder to which the ureteral catheters are anchored. These are allowed to remain in place several days so that ureteral edema may subside before their removal.

Occasionally, a second and even a third attempt is made to engage a stone transurethrally. At times, when manipulation has not been successful, and a high degree of obstruction does not exist, one may be content to dilate the intramural ureter as well as the remainder of the ureter distal to the stone, using the Councill stone extractor, with expectation that the calculus will pass spontaneously from the ureter within a week or so after dismissal from the hospital. This approach is not nearly so gratifying to the patient and physician alike as that which results in delivery of the stone into the hands of the family immediately postoperative.

In the event that a high degree of obstruction persists, after an unsuccessful manipulative effort, and should one then be unable to pass the point of obstruction with a ureteral catheter in order to establish drainage, ureterotomy then should be

done rather promptly. The possible presence of contamination proximal to the obstructive point, coupled with the factor of a closed space poses a distinct emergency. There is no problem in urology requiring more prompt attention than a high degree of obstruction accompanied by infection. Failure to employ immediate surgical intervention may result in loss of a kidney or even in death.

In absence of appreciable obstruction one may allow the wishes of the patient to influence the action to be taken, only however, after the various contingencies are fully understood by all parties concerned. This is particularly desirable when the calculus stands a fair chance of passage to the lower ureter. Pressing matters of business or the desire to prevent expense incidental to hospitalization and surgery occasionally enter the picture. Often enough, one is able to spontaneously pass a fairly sizeable stone without resultant damage. Yet, frequently, if the calculus is not passed, days even weeks may be consumed with loss of work and unnecessary endurance of pain only to accept, at a latter date, definitive treatment which should have been actively instituted at the outset. In spite of this, there will always be those who seem bent on convincing themselves first as to the need for action before submitting to prompt management.

One should not allow the wishes of the patient to influence the proper course of action when a high degree of obstruction exists. Needless to say, this problem does not present very often, for the severity of the pain is commonly too great to permit dilatory action.

Success with the so-called antispasmodic drugs in effort to assist passage of ureteral calculi has not been impressive in my experience. These may well be used, however, where expectant management has been chosen.

COMPLICATIONS

Complications attendant to open surgery are those commonly seen where excessive trauma to a ureter has resulted either through "hard luck" surgery or poor management of the problem postoperatively. Great care should be taken to prevent the development of ureteral stricture. Ureterotomy drainage, as with a gall bladder T-tube, is not employed owing to the likelihood of subsequent stricture formation. If one must splint the ureter it is best done by passing the supportive tube up the ureter and down into the bladder removing it transurethrally at a later date. A water tight closure of the ureter, in fact an attempt at closure at all, is rarely undertaken.

Manipulation of a calculus may result in ureteral perforation which need not cause much concern if recognized and the problem at hand carefully considered. Exploration and drainage of the perforated site is only occasionally necessary.

A cuff of the ureter may be pulled into the bladder—a complication too which is not pleasant to behold but again need not be attended by much excitement, particularly if catheters can be passed to the renal pelvis allowing drainage for a few days.

Loss of a filiform tip or even the entire basket portion of the instrument, in the ureter, may be troublesome; however, I have seen these pass spontaneously. Reverse peristalsis may carry these foreign bodies to the renal pelvis necessitating a pelviotomy. Exploration of the lower ureter may be required, or with fortune, one may manipulate, engage, and remove the foreign body transurethrally.

It is well for all patients who have had ureteral calculi removed, by whatever means, to have an excretion urogram made within six weeks to three months following the effort. This time interval varies in accordance with the circumstances attendant to the particular case in question. Similar examinations, over a longer period of time may also be in order. In this way one may prevent and forestall circumstances which could ultimately lead to renal destruction.

Effort should be directed to the prevention of urinary calculi in order to remove

the vicious triad of obstruction, infection, and destruction with which we as urologists constantly do battle. Granted that infection and obstruction have been eliminated there is possibly no factor more important in stone prevention than an adequate fluid intake. Admittedly, there are certain exceptions common to each problem, but in the main, a fluid intake in excess of 3000 cc. daily will do as much as most any other measure to assist these patients in the prevention of urinary calculi.

CONCLUSION

There are few problems with which the urologist is confronted which are more taxing on his professional ability and ingenuity, and none in which greater pleasure is experienced by patient and physician alike than the successful and proper management of a ureteral calculus.

DISCUSSION

Dr. Thomas Latiolais, Jr., (Lafayette): This will be a reiteration rather than a discussion as the entire presentation was very well put, and to the point.

I think that the patient suspected of having a ureteral calculus, regardless of the duration of symptoms or his general state of health, should have a scout film of the kidneys, ureters and bladder and an excretory urogram prior to instrumentation. A diagnostic or operative cystoscopy should be avoided until a definite plan of management is attained. Many times this procedure eliminates instrumentation completely or allows the urologist more time to better evaluate the best procedure for the individual case.

The old dictum, "Stones less than one centimeter in diameter will pass," does not seem to hold true when they are found in the upper ureter, particularly, when the calculus presents irregularity in any degree. There is usually less danger, less expense, less instrumentation and the patient is returned to his normal activity in a shorter period of time, when the muscle splitting operative procedure is done.

Because of the technically more difficult operative procedure, longer morbidity, and accesibility, stones in the lower ureter present a little different situation. Transurethal manipulation is more often the procedure of choice, even with the larger calculi up to 1.5 centimeter in diameter. I find that an adequate ureteral meatotomy allows insertion of ureteral catheters more readily immediately after stone extraction.

At this point I think it advisable to mention "the good old nephrostomy operation" which has pulled me out of a good deal of trouble following unsuccessful transurethral manipulations accompanied by fulminating infection behind the calculus. The peristaltic action of the ureter is still intact when a nephrostomy tube is in place and the smaller remaining calculi will eventually pass spontaneously.

I would like to congratulate Dr. Doss on his excellent, concise discourse and I am really honored in giving this discussion.

CONSIDERATIONS IN BILATERAL CRYPTORCHISM*

CHARLES A. HOOKS, M. D.†
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That my subject is not a new one is illustrated by a quotation from the 12th verse of the 19th Chapter of St. Matthew which says, "For there are some eunuchs which were so born from their mother's womb—". Presumably, this includes what we now designate as bilateral cryptorchism or perhaps other alterations of the somatosexual pattern in which the testes are absent from their customary location.

The manifestations are so varied that I find it helpful from a clinical standpoint to consider the problem in a broad way, incorporating:

1. Pseudocryptorchism (migratory testes in infants)
2. Endocrine disorders with imperfect testicular descent (enunuchoids, etc.)
3. Monorchism or anorchism (congenital absence of one or both testes)
4. Ectopic testes (a deviation in descent)
5. True cryptorchism (arrest in normal descent)
6. Intersexuals (altered somatosexual pattern)

In the first four groups, a more or less definitive approach and management has been attained; whereas the latter two entities are complex, for there is still much speculation based on incomplete evidence. The challenge is more intriguing when

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there seems to be imperfect descent of both testes or when there are complicating genital anomalies.

The prime aim in treatment of cryptorchism is preservation of the gonadal function. Irreversible destructive effects are known to occur in the tubular germinal epithelium of the undescended testis which is retained into the pubertal and postpubertal periods. For this reason, and since definition of puberty is uncertain, there are some who now urge completion of endocrine or surgical treatment or a combination of both before the child is five years old. On the other hand, a majority of uncomplicated cryptorchid testes, it is said, descend spontaneously during normal pubertal transformation, and yet it appears that we do not know to what extent the fertility of these individuals may have been impaired.

Errors in judgement or surgical technique may not only nullify the spermatogenic potential, but may actually injure the patient's subsequent mental and physical well-being. Consideration must be given the frequent anomalous separation of the epididymis and vas deferens from the testicle which interrupts the spermatozoal conduit. Moreover, Warren O. Nelson has recently demonstrated by simultaneous biopsy of both testes in unilateral cryptorchids that spermatogenic defects of a constitutional nature affecting both testes are sometimes associated with cryptorchism This is the same sort of disorder encountered in certain infertile men with normally descended gonads.

While much helpful data has been accumulated, the confusion that exists generally is expressed in the lack of agreement as to timing and application of treatment. Only a standardized evaluation of all methods in many hands over a sufficient period of time for adequate follow-up can provide a basis for more consistent premise. It seems logical then to ask how often we can expect our currently prevailing treatment programs to actually preserve spermatogenesis.

The tendency for malignant neoplasm to develop in the cryptorchid testis either before or after orchidopexy is recognized, although there are some who feel that this point has been overemphasized as an argument for early reduction of the testis to scrotal position. Some patients seek treatment to improve their "shower room" appearance or to advance their economic status through compliance with the requirements of employment programs. Repair of the inevitable hernia is accomplished, and the likelihood of torsion of the spermatic cord is lessened. In addition, the testicle is usually placed in a more favorable location to avoid trauma.

Increased pituitary activity reflected by elevated urinary gonadotropin excretion in the postpubertal cryptorchid is evidence that the interstitial tissue also suffers. However, the clinical effects are seldom discernible, and alterations in the histologic picture are slowly progressive. Hence, consideration of this aspect is relatively unimportant.

Our most interesting experiences have occurred in the realm of the intersexes where one finds variance in morphological and germinal sex expression. We were precipitated into a study of this entity in the summer of 1947 by the rapid-fire referral from our Pediatric Department of two "male" infants with hypospadias, bilateral cryptorchism, and clinical evidence of cortical hypoadrenalism. Urologic study, including abdominal laparotomy and gonadal biopsy, proved that these were both genetic females. Excessive androgenic activity of the fetal adrenal cortex during the critical period of sex differentiation resulted in formation of external genitalia of male type while the internal structures were typically female. These were female pseudo-hermaphrodites.

It is thought that through other ill-defined genetic and hormonal influences during intrauterine life male psuedo-hermaphrodites are produced. These individuals have external genitalia resembling a female and gonadal tissue of a male.

True hermaphrodites, sometimes called gonadal intersexes, are thought to result

from genic mutations. They are less commonly encountered and present a variety of alterations in somatic structure as well as mixed pattern of the germinal tissue. As a rule, both testis and ovary are found in the same gonad; however, in rare instances, one may find ovary on one side and testis on the other. There are two such "gynandromorphic" hermaphrodites in our present study. Two others are known to possess ovotestes.

Regardless of the prenatal cause of such postnatal anatomical abnormalities, one is always faced with a series of difficult decisions in reaching an acceptable solution of the problem. Even legal technicalities must be dealt with in the correction of birth certificates in instances where the wrong sex has been mistakenly assigned at birth.

The physician must be alert to the possibilities as soon as he inspects the newborn babe. It is my opinion that all hypospadias must be viewed with suspicion and investigated for significant evidence of intersexuality. Such need is more urgent when there is an associated bilateral "cryptorchism."

Even in infants appropriate urethrocystoscopic examination can be accomplished. Varying degrees of exaggeration of the utricle may be observed; and, indeed, in some it represents the vaginal orifice. If the cystoscope sheath can be inserted, the interior of the vagina is inspected and cervix identified.

Roentgenographic demonstration of the rudimentary vagina and, in some, the cavity of the uterus and fallopian tubes is often helpful. Now and then, we see signs of spilling of the radio-opaque medium into the peritoneal cavity. For this examination we have preferred to use a 15 to 20 per cent aqueous solution of Skiodan injected through the cystoscope sheath, a soft rubber catheter, or directly with an Asepto syringe as the occasion demands.

Abdominal laparotomy must be done to determine the status of the internal genital structures and to identify the type of germinal tissue. Longitudinal axial biopsy is essential since there may be bi-polar accumulation of ovary and testis. Histologic study of serial sections is often tedious and time consuming; for example, Mrs. Octavia Hall, a Research Associate in Physiology at the University of Texas prepared and examined some 300 serial sections of gonadal tissue submitted from only one of our "gynandromorphs."

Having appraised the abnormal somatosexual pattern, a decision must be made in selecting the most appropriate form of readjustment. Although gonadal sex may be known, the general sex-direction of the body tissues as a whole is not usually apparent during the prepubertal phase. Delay in beginning treatment until after puberty often permits development of other difficult problems particularly in attitudes of the patient or his associates. Work is currently underway in the University of Texas Tissue Culture Laboratory directed by Dr. C. M. Pomerat in cooperation with Dr. T. C. Hsu through which we hope to be able to accurately determine the tissue sex of these patients even before gonadal biopsy is done. This accomplishment would be of infinite value since it would permit the earliest possible anatomic reconstruction, psychotherapy, and sociologic adjustment, followed later by well-founded endocrine substitution therapy should that prove necessary. Thus there would seem to be optimum opportunity for these unfortunates to become acceptable members of the society in which they must live. Notoriety sometimes complicates delayed adjustment. An extrovertive attitude which capitalizes on tragedy is rarely encountered in the true intersex. More often these patients are introvertive and depressed and occasionally develop suicidal tendencies.

The physician's burden of responsibility is great in these cases of real or apparent failure of the testes to descend. He must determine whether simple testicular retention or sexual dimorphism is represented. An error in decision may prove costly to fertility in the first instance or in the lat-

ter, where fertility is not a problem, may interfere with optimum sociologic adjustment. Despite incomplete data, the present trend indicates that better results may be obtained if treatment is instituted early in the prepubertal period.

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PIROMEN THERAPY IN DERMATOLOGY

A CLINICAL EVALUATION OF 1,029 CASES

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NEW ORLEANS

Dermatologic conditions historically have presented physicians with a stubborn problem which often resists conventional forms of treatment. In recent years several new therapeutic agents have been developed which have increased the physician's effectiveness in treating such disorders. Piromen, † a relatively new therapeutic agent, shows promise of being particularly valuable in treating certain forms of dermatitis and may be added to this list.

Piromen is a biologically-active, complex polysaccharide prepared by chemically fractionating cellular material derived from a *Pseudomonas* species. It is nonprotein, nonantigenic, nontoxic, and may be employed safely within an extremely wide dosage range. It is prepared in stable colloidal dispersion for parenteral use. [1]

Injected in proper dosage, Piromen produces a marked neutrophilic leucocytosis, usually within four to six hours.[2] It also stimulates the reticuloendothelial system, and there is evidence of the stimulation of the reticular zone of the adrenal cortex.[3]

There are few and relatively minor undesirable effects from piromen. Occasionally, if dosage has been sufficiently high to cause a pyrogenic reaction, a patient may experience chills and fever, moderate headache, myalgia, or nausea. Aspirin will relieve much of the discomfort.

The literature on previous clinical studies utilizing piromen indicates that the new drug has distinct value in treating allergy and dermatitis.[4-10] There is evidence that many dermatologic cases have a definite allergic etiology, and that in spite of antiallergic management such as diet, desensitization, or avoidance of inhalants, some patients do not obtain satisfactory clinical relief. Piromen therapy is recommended for such patients.

Dermatologic conditions vary in severity and in response to treatment from patient to patient. The success of the treatment depends on the psychological and general make-up of the patient as well as on the therapeutic value of the treatment. Thus, it is possible in some cases to obtain a satisfactory response with the simplest of medication, while in other cases satisfactory results are not forthcoming, no matter what the treatment.

For this reason it is difficult to evaluate the efficiency of any therapeutic agent unless sufficient cases are considered to allow for this human factor. This study is an evaluation of 1,029 cases involving 84 varieties of dermatitis which have been treated over the past eighteen months.

DISCUSSION

In all cases piromen was given as an adjunct to usual topical therapy. Individual dosages varied from 1/2 gamma (microgram) to 6 gamma and the duration of treatment from one visit to nine months of regular weekly visits. The number of injections varied from 1 per patient to 45, the total quantity of the drug from 1 gamma to 116 gamma, and the age of the patients from 2 months to 89 years.

A summary of the disorders involving 79.5 per cent of the total cases is shown in Table 1. The remaining 20.5 per cent is too widely scattered among a variety of dermatologic forms to be clinically significant.

The classification remission includes

* Professor of Dermatology, Louisiana State University School of Medicine, at the time of his death on April 2, 1953. This paper was to have been presented at Annual Meeting of the Louisiana State Medical Society, May 7-9, 1953.

† A product of Travenol Laboratories, Inc., a subsidiary of Baxter Laboratories, Inc., Morton Grove, Illinois.

TABLE 1.

Diagnosis	Total Cases	Remission	Improved and Discontinued	Unimproved and Discontinued	Improved and Still Treating	Unimproved and Still Treating	Discontinued*
Vesicular Dermatitis †	55	35	5	3	8	1	3
Otitis Externa	35	17	3	1	7	2	5
Seborrhoic Dermatitis	94	37	10	3	22	2	20
Acne	120	24	27	6	44	3	16
Contact Dermatitis	173	93	28	6	23	2	21
Pyodermia	120	67	8	1	27	–	17
Leucoplakia	18	2	1	2	8	5	–
Onychomycosis	27	1	5	2	12	2	5
Tinea	126	44	23	6	33	3	17
Pruritis Ani	5	2	–	–	3	–	–
Pruritis Vulvae	7	3	–	–	3	1	–
Erythema Multiforme	9	3	4	1	1	–	–
Uriticaria	12	4	2	–	1	1	4
Lupus Erythematosus (Discoid)	17	1	–	2	8	2	4
TOTALS	818	333	116	33	200	24	112

* Discontinued after first injection. Patient did not return—reason unknown.

† Includes: contact dermatitis which was vesicular; pompholyx; epidermophytosis; and bacterid, vesicular in type.

those cases discharged as cured and those essentially well but who failed to return for additional check-ups. Those listed as discontinued for the most part were patients who were seen once and did not return for further visits.

The category vesicular dermatitis includes: contact dermatitis which was vesicular; pompholyx; epidermophytosis; and bacterid, vesicular in type. Contact dermatitis not vesicular is classified separately. Seborrhea or seborrhoic dermatitis confined to scalp only is classified as seborrhoic dermatitis.

In the beginning of this study piromen was injected intravenously. However, several patients had febrile reactions with nausea which continued for several hours, so intravenous injections were discontinued. Those cases are not included in this study.

Thereafter, patients were given piromen intramuscularly or subcutaneously. A routine therapy was 1 gamma on the first injection and 2 gamma on subsequent injections. At this dosage only one patient had a reaction. She had severe chills and nausea after the first injection of 1 gamma and after the third injection of 2 gamma. The dose subsequently was reduced to one gamma intramuscularly and she had no further reaction.

One other patient claims she had a flare-up after receiving piromen. It is doubtful that piromen was the cause because she did not have a marked febrile reaction. However, she was excluded from further piromen therapy.

For the most part, patients reported a feeling of well-being after an injection of piromen. Their discomfort seemed to decrease and there was accompanying improvement in their mental attitudes. This has been reported by other investigators as well.[4, 11-13]

CONCLUSIONS

The clinical study indicates that piromen is of great benefit in treating a variety of dermatologic disorders, particularly otitis externa. Twenty-seven of 35 otitis externa cases, the majority of them old cases which had failed to respond satisfactorily to other treatment, showed remission or definite improvement after piromen therapy. The results in 5 other cases are unknown and listed as discontinued because the patients failed to return after the first treatment.

H. G., 51, female, had been treated for an otitis externa since June 1948, with x-ray, local applications, and injections of calcium gluconate

and mixed vaccine, without particular benefit. She was started on piromen April 14, 1952, with a 1 gamma injection, then received 2 gamma weekly for five weeks. Considerable improvement was noted after four weeks. She was then given 2 gamma of piromen every second week until August 21, and has had no further complications since that time.

U. R., 58, female, had a severe otitis externa and an eczematoid dermatitis of the ear lobe. Her ear ached constantly. Prior treatment with topical agents had produced little result. She was put on piromen (1 gamma initially) and given 75 r x-ray into the ear canals. Pain disappeared after the first injection of piromen and after a series of 2 gamma injections she was symptom free and required no further therapy.

R. L., 40, female, had an otitis externa of fungal origin. She had been treated since February 23, 1949, by x-ray to the ear canal and with boric acid and alcohol earwash with little result. In October 1951, she was placed on piromen therapy and 2 gamma injections were given every two weeks. The otitis externa was kept under control but did not clear up completely.

Vesicular eruptions, seborrhoic dermatitis, and tinea also responded more quickly under piromen therapy than would have been expected without the use of the new agent. While the value of piromen is not as clearly defined in these cases as in otitis externa, there is no doubt that it definitely improved the therapy.

R. B., 29, female, reported a long period of vesicular eruption of the hands. She was started on piromen, November 2, 1951, with a 1 gamma injection and then received 2 gamma weekly for nine weeks. Improvement was apparent after the first week, and after five weeks her response was extremely gratifying. The patient's hands were clear by January 24, but she was continued on a maintenance dose of piromen, 2 gamma every other week, until March 27, when treatment was discontinued.

J. D., 24, female, had a long history of vesicular eruption of the hands. Prior treatment elsewhere had produced no results. She was started on piromen, November 14, 1951, with one gamma. The dose was increased 1 gamma each week until she was getting 5 gamma weekly. This continued for five weeks, at which time the dose was reduced to 2 gamma weekly and then to 2 gamma monthly. She received thin window light treatment as well. The patient responded well to the treatment until she neglected to return for further treatment. She then had a mild flare-up which again cleared nicely after injections were resumed.

L. B., 63, female, suffered severe itching from seborrhea of the scalp. She also had an otitis externa. She was first seen June 27, 1950, and treatment for a year with x-ray and local ointments produced little improvement. Piromen was added to the therapeutic regimen April 1, 1952, and she received regular 2 gamma injections. She was last seen November 4, 1952, and was very much improved.

C. H., 18, female, had an "id" infection of the hands of lineal origin and fungus of the feet. She was placed on piromen April 26, 1952, with a 1 gamma injection and then was given weekly injections of 2 gamma for four weeks. She showed much improvement after three injections and a week later her hands and feet were clear.

It is more difficult to evaluate the role of piromen in contact dermatitis. Both remissions and exacerbations were noted. Piromen was used in conjunction with topical therapy in all cases and occasionally was given in conjunction with cortisone. However, there were sufficient helpful results from piromen to merit its continued use as adjunct therapy. The same is true of cases of pyodermia, most of which received chemotherapy as well as piromen.

E. C., 31, female, had an early case of contact dermatitis of the hands. Treatment was begun October 11, 1951, with topical applications, x-ray, and injections of calcium gluconate. Piromen was given December 7, with a 1 gamma injection the first week, 2 gamma the second week, 3 gamma the third week, and then the dose was reduced to 2 gamma weekly. Her response to treatment was very good after piromen was given and after three weeks her hands were entirely clear. She was continued on a maintenance dose of piromen until April 2, 1952. She has had no recurrence.

Piromen therapy does not appear very promising in cases of psoriasis, vitiligo, or alopecia. The bulk of the unimproved cases fall in these categories. Neither is the role of piromen apparent in cases of acne. I still use some piromen in treating acne but only as a treatment aid and not with the idea of clearing up the condition through piromen therapy.

SUMMARY

1. A group of 1,029 patients suffering from 84 dermatologic disorders received as adjunct therapy injections of piromen —a nonprotein, nonantigenic, *Pseudomo-*

nas polysaccharide prepared from bacterial cellular material.

2. Piromen proved excellent for treating otitis externa and of definite value in vesicular eruptions, seborrhoic dermatitis, and tinea.

3. The value of piromen in treating contact dermatitis and pyodermia is difficult to evaluate, but there are sufficient indications of its worth to merit its continued use.

4. Piromen therapy does not appear promising in cases of psoriasis, vitiligo, or alopecia, and is of indefinite value in treating acne.

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AN EFFECTIVE METHOD OF PERSONAL ORAL HYGIENE*

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NEW ORLEANS

INTRODUCTION

The purpose of personal oral hygiene (usually called home care of the teeth) is (1) maintenance of oral cleanliness, (2) prevention of caries, (3) prevention of periodontoclasia. It is desirable not only to prevent initiation of the lesions of these diseases, but also to prevent, to the maximum extent possible, further advancement of lesions that already exist. An effective method must fulfill all three of these important purposes.

A clean tooth does not decay. Periodontoclasia does not occur about a clean tooth. Therefore, a method which satisfies well the first purpose named, at the same time accomplishes the other two.

No amount or quality of treatment and restorations by dentists can maintain dental health unless they are followed by effective personal oral hygiene. The success and durability of the dentist's work depend largely upon the subsequent personal oral hygiene of each patient.

Of the many methods of personal oral hygiene now generally followed, none is entirely effective. This statement is confirmed by the fact that practically all adults, and many younger persons, have already sustained more or less damage from caries, and that periodontoclasia lesions are present around many or all of the teeth of practically all adults. This means that the methods of personal oral hygiene generally followed, whatever the source of instruction, are inadequate to prevent these diseases.

Several years ago I designed and described [1] the method of personal oral hygiene which is necessary for prevention of caries and periodontoclasia. It was based upon accurate information, secured mostly through laboratory research, upon the etiological conditions at the locations where the lesions of these diseases originate and advance. Measures for successful prevention must provide for effective prevention or minimizing of these local etiological conditions. Correct information (not merely opinions) as to what these conditions are is necessary. Measures based upon incorrect information, erroneous beliefs,

* Studies promoted by facilities to which the author has had access at the School of Medicine, Tulane University of Louisiana, and by aid for equipment and supplies provided by the University.

or misinterpretation of scientific observations, however authoritative they may seem to be, are unlikely to be effective.

The causative organisms in these diseases are microscopic, the pathological processes originate and advance microscopically, the tissues involved are composed of microscopic elements and the lesions themselves, at first, are only microscopic in extent. Therefore, correct information about these conditions must be based upon microscopic research. Opinions and ideas not conforming to the basic facts, which can be confirmed only by microscopic work, are confusing, often entirely erroneous.

During the past several years there has been opportunity to demonstrate, to their satisfaction, and to teach to a considerable number of dentists, the fundamental facts as to the local etiological conditions in these two diseases. With these facts known and understood, and any previously existing confusion or conflicting ideas cleared up, it is evident to anyone that effective personal oral hygiene is necessary for prevention of these diseases; and to significantly retard or prevent further advancement of existing lesions. It is also evident that methods generally taught and practiced are inadequate and could not be entirely effective.

The necessary method, which I have described,[1] is quite different from, and in some particulars quite the opposite of, ineffective methods generally advocated and followed. Dentists who have the necessary information have their expectations confirmed and are profoundly impressed by the effect they themselves experience personally, and the beneficial effect upon their patients whom they successfully teach. Hundreds of people (including a good many dentists) can be named who now, by this method, are maintaining a degree of oral cleanliness and dental health far greater than is possible in any other way.

The purpose of this paper is again to direct attention to this method, I now having had considerable experience in presenting the fundamental facts upon which it is based, and in teaching the method to a considerable number of people (mostly university personnel). But before describing the method it seems desirable to recall briefly or present important information about the local etiological conditions which must be minimized or prevented and to recall or present some of the fundamental facts regarding the disease processes involved.

Clear understanding of the cause of a disease is necessary for the adaptation of means for its prevention. If the disease be local, such as caries and periodontoclasia, clear understanding of the essential local conditions which cause the lesions and promote their advancement, is necessary for adaptation of means of preventing these local conditions.

BASIS FOR THE METHOD

Locations where enamel caries lesions originate.—Caries lesions of the enamel first appear in the occlusal pits and fis-

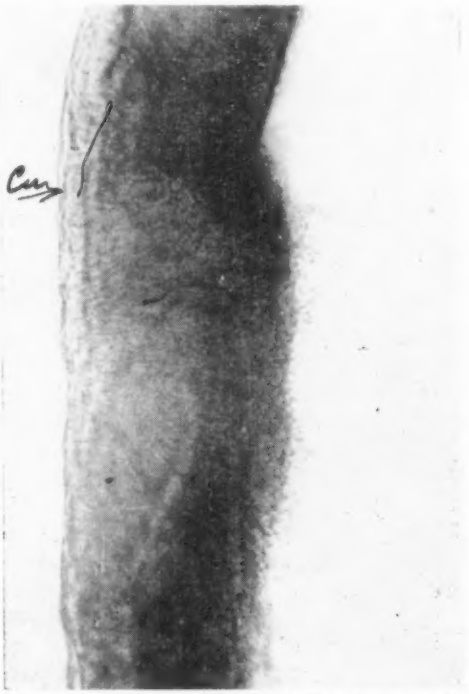

Fig. 1: Section of bacterial film from surface of enamel. Enamel cuticle, cu, at left. Note fruiting heads projecting at surface of pad at right.

sures and on the proximal surfaces between the teeth, at locations where there is more or less protection from natural cleaning. They do not originate on surfaces from which foreign material is frequently removed by functional friction or otherwise.

Nature of the material in protected areas.—In uncleaned areas on the teeth there is continuously present a heavy film (Fig. 1) of bacterial material (usually called plaque) consisting of long rod and filamentous types of microorganisms, characterized by having one end attached to the tooth (Fig. 2) and the other extending outward to the surface of the film where there are the growing ends and

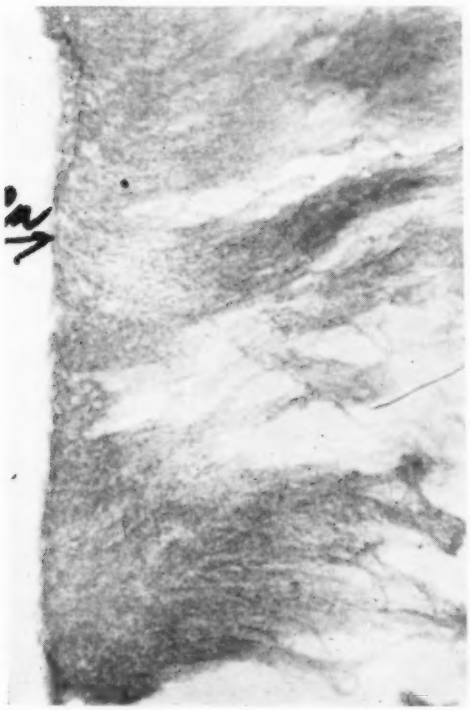

Fig. 2: Section of bacterial film from surface of enamel slightly teased apart to show filamentous type organisms. Enamel cuticle, cu, at left.

usually fruiting heads of the fungi making up the pad. The thickness of this bacterial film varies greatly at different locations, determined largely by the depth of the space and by the exposure to friction by which the ends of the filaments at the surface are frequently rubbed off. At the surface there are not only the growing ends and fruiting heads of the filaments making up the pad but also large

Fig. 3: Bacterial film from over active chalky enamel. Outer part of film brushed off leaving stumps and stems of filamentous type organisms attached to cuticle, cu.

numbers of the very many varieties of other bacteria in the mouth. These are accumulated upon the surface and interspersed, more or less, between the outer ends of the filaments.

Deeper in, however, the pad at any particular place is made up of the filamentous types only, each filament extending outward, at right angles, from the surface of the tooth to which it is attached. Breaking off the ends of the filaments at the surface of the pad still leaves a film consisting of stumps and stems (Fig. 3) attached to the tooth. Most of these are living and capable of growing in length again under favorable circumstances, thus restoring, in time, the former thickness of

the film pack at any given location.

The presence on teeth of filamentous types of microorganisms, usually spoken of as Leptothrix or thread forms, has been long recognized, some authors associating them with caries, others not. More than eighty-five years ago Leber and Rottenstein [2] described filamentous forms of Leptothrix as the "microorganisms of caries." Their Figs. 1, 2, 3 in Plate II, illustrate the organisms and a pad of filamentous forms attached to, and radiating from, a decaying tooth surface.

Wedl [3] in 1870, emphasized the proliferation of Leptothrix in relation to caries and illustrated this in several of the engravings in his *Atlas to the Pathology of the Teeth*, showing uniformly disposed, firmly adherent Leptothrix masses on exposed surfaces of teeth, especialy those at which caries lesions originate.

W. D. Miller [4] in 1883, observed filamentous forms of Leptothrix on the teeth and in caries lesions. He describes, on the border of decaying dentin, indistinguishable masses of fungi from which project numerous threads of *Leptothrix buccalis* (See his Fig. 2). Later, in 1890, in his book [5] on *The Microorganisms of the Human Mouth*, his Figs. 63, 67, 79, indicate filamentous organisms composing the bacterial film on teeth. He was not able to identify these filamentous forms, or any other forms of bacteria found in the mouth, as the specific cause of caries. He evolved his well known chemicoparasitic theory to the effect that acids are produced by the breakdown of carbohydrates by acidogenic bacteria at the vulnerable locations; which acids decalcify tooth material and thereby cause and promote caries. All firm information to this day confirms and supports the correctness of this theory formulated more than sixty years ago.

In a recent survey of the literature relative to the cause of caries Boedecker [6] came to the general conclusion that "intact enamel surfaces in vitro exhibit widely variable resistance to attack by acids produced by acidogenic bacteria. There is no evidence that acidogenic bacteria and carbohydrates can initiate dental caries". (I do not accept this conclusion. Acidogenic bacteria and carbohydrates are essential for the initiation of caries lesions). "The progress of dental caries, once initiated, is effected by bacteria acting during the acidogenic phase on a carbohydrate substrate." "There is probably no specific microorganism associated with dental caries . . ."

The conclusions reached by Bartels [7] from a survey of the recent literature on the subject do not conflict with or contradict Miller's [5] original theory, but rather support it.

The fact that bacterial action on cariogenic food material within the mouth is necessary for production of caries is further substantiated by the work of Kite, Shaw, and Sognnaes [8] who showed that susceptible rats fed a cariogenic diet by stomach tube do not develop caries; and by Orland [9] to the effect that rats maintained entirely free from bacteria, reared upon a cariogenic diet, do not develop caries, whereas controls on the same diet have a high percentage of caries.

The work of J. Leon Williams, toward the close of the nineteenth century, contributed more than all others had previously, to the cause of caries and the microscopic conditions at the locations where the lesions originate. His paper [10] in 1897, not only shed much light upon the microscopic caries process, but it showed clearly the nature of the bacterial film upon the surface of teeth beneath which the caries lesion originates and advances. In numerous photomicrographs (See his Figs. 61, 62, 63, 64, 66, 67, 69, 71, 75) he showed beyond question that the film pack is composed of long filaments radiating outward from the surface of the tooth to which they are attached. His work substantiated the claims of Miller [5] and of G. V. Black [11], that caries is caused by acids produced by conditions at the location of the lesions, and showed the morphologic characteristics of the principal microorganisms composing the film pack.

Later, Williams [12] directed attention to the numerous microorganisms found in material scraped from the surface of the teeth, some of them long rods and filaments, together with many other forms—large and small rods, and coccal forms. He directed attention especially to an organism which had been described previously by Vicentini [13] and named by him *Leptothrix racemosa*. This organism consists of a long relatively coarse filament or stem, attached to the tooth, on the outer portion of which numerous spores are produced. This spore-bearing conidiophore, which they called a fruiting head, is found projecting at, and just beyond, the surface of the film pack.

The striking, characteristic fruiting head serves as a convenient means of identifying the organism. Although it contributes largely to the bacterial film on many teeth, there is no evidence that it bears any specific relationship to caries. However, it is usually one of the several thread-form and filamentous type organisms making up the film on teeth, beneath which caries lesions originate and advance. In many specimens the deeper part of the film is made up largely of the stems of this fungus.

Vicentini [13] claimed that it is present in all mouths. Williams, in his first paper describing it [12], states that it "is common to all mouths which I have examined." I have found it present in material from most of the hundreds of extracted human tooth specimens, from many different sources, which I have examined.

The morphologic characteristics of this organism and the way in which it often makes up a large part of the film pack are shown by Williams [12] by numerous photomicrographs. (See his Figs. 14, 17, 18, 20, 31, 32, 33, 34, 37). I have also shown [1] (See my Figs. 6, 7, 8, 9) how the fruiting heads of this organism tend to project above the surface of the film pack and to accumulate in masses at the surface.

Throughout the literature since the historic paper of Williams [10] in 1897, there may be found numerous pictures of preparations which confirm the fact that the bacterial film on teeth is composed largely of long filamentous forms, each one extending outward from the surface to which they are attached. Since an effective method of personal oral hygiene must be based upon correct information as to the characteristics of this bacterial film, it may be helpful to refer to a few of such illustrations: [10] (Figs. 6, 8, 9, 11, 24), [11] (Figs. 2, 3), [12] (Figs. 82, 84, 109), [13] (Figs. 3, 4), [14] (Figs. 86, 87), [1] (Figs. 4, 5, 6, 7, 8,), [15] (Fig. 3), [16] (Fig. 3), [17] (Fig. 3, 4), [18] (Figs. 86, 87), [19] (Fig. 2), [20] (Fig. 3).

Conditions within cavities.—If the conditions on the tooth which initiate and promote the first stage, chalky enamel, caries lesions continue long enough, sooner or later a cavity develops and advances, usually at an accelerated rate. Active cavities in enamel finally reach, and advance into the dentin; thus progressively increasing in depth and in width.

Cavities, and to a less extent normal pits and fissures, present favorable receptacles for lodgment and retention of food material. The larger and deeper the cavity, the more food material it will hold. Many cavities are never free of remnants of food, and this material is continuously in various stages of decomposition. Before lodgment in the cavity food is heavily inoculated (or contaminated) with the many different kinds of bacteria [5, 12, 20-23] of the oral flora. In the cavity it is exposed to the abundant transient bacterial flora there, arising from decomposition that is going on of previously introduced food material, and also to the more or less permanent or continuous bacterial film lining the base and walls, (Figs. 4, 5) of the cavity.

Such a conglomerate bacterial environment always contains many species which produce acids when growing in the presence of fermentable carbohydrates, and others which do not. Food material (particles) retained within a cavity may, and usually does, contain variable amounts of

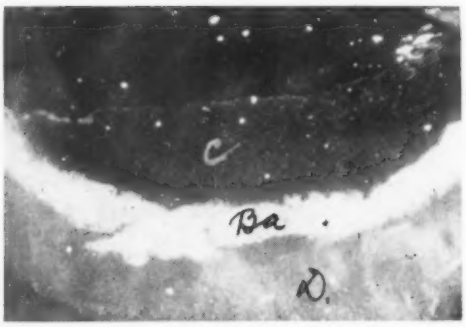

Fig. 4: Section including bottom wall of cavity in dentin, photographed with incident light. Ba-bacterial film, D-dentin, C-remnant of celloidin in which specimen was embedded.

Fig. 5: Section of sidewall of cavity in dentin showing filamentous bacterial pad at extreme right, disintegrating dentin to which film is attached, and spindle shaped expansion of tubules packed with bacteria.

such carbohydrates. In addition to that present in the particular material when it was introduced into the cavity, the carbohydrates may be replenished, more or less, from carbohydrate containing fluids (drinks) taken into the mouth subsequently. The production of acids, upon which caries activity and enlargement of the cavity depend, is determined largely by the retention and break-down of fermentable carbohydrates within the cavity.

The nature of the more or less continuous bacterial film lining the walls of the cavity is of great importance, from the standpoint of oral hygiene for prevention of further advancement of the lesion. This film is made up of countless millions of filamentous type organisms (Fig. 6) similar to those making up the film on the surface of teeth beneath which caries lesions originate. One end of the filament (Fig. 7) is attached to the decaying walls of the cavity, from which it extends outward to the surface of the film, where there are the growing ends and fruiting heads.

Fig. 6: Section of bottom wall of cavity in dentin. Deeper part of bacterial film consists of filamentous type organisms attached to decaying dentin.

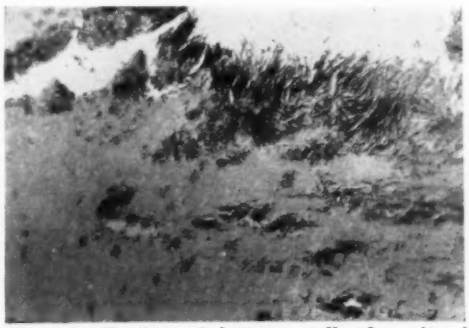

Fig. 7: Section of bottom wall of cavity in dentin. The outer part of the bacterial film has been torn away leaving stumps and stems of the filamentous type organisms attached to the disintegrating dentin.

The thickness of this bacterial film within the cavity varies greatly, depending largely upon the dimensions of the cavity and how frequently and completely its contents are disturbed or removed. Many of the films within cavities in extracted tooth specimens which I have measured range from about 100 to 400 microns (Fig. 8); some considerably thicker. Most of the filaments of the different kinds of organisms composing such films are less than 0.5 micron in thickness. Therefore, the attached film within the cavity is made up of millions of separate elements, each of which is many times—usualy several hundred times—as long as it is thick.

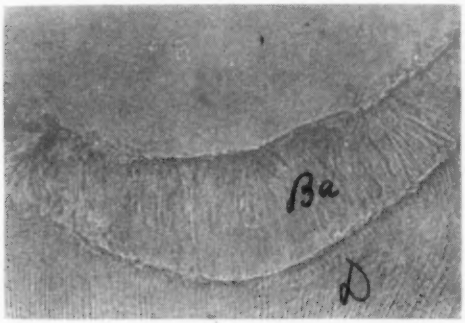

Fig. 8: Heavy bacterial film, Ba, lining bottom wall of cavity in dentin, D. Thickest part of film pad measures 430 microns.

Every specimen of decaying teeth shows precisely the same thing. Covering the surface or lining the cavity where decay had commenced there is always to be found a thick, felt-like mass of filamentous types of organisms, each radiating outward from the surface to which it is attached.

Pertinent facts relative to the caries process in enamel.—If a tooth specimen is suspended in a strong solution (5 to 10 per cent) of HC1, the enamel is rapidly dissolved. The enamel cuticle is set free within a minute or two, by the dissolving of the enamel immediately beneath it, and this loosened keratin-like membrane now may be removed or brushed off. If the acid is greatly weakened by sufficient dilution, or by less dilution plus the addition of tribasic calcium phosphate, then decalcification proceeds very slowly; and in the latter case only the carbonates are removed at first [24, 25]. Only partial decalcification occurs. The enamel becomes porous and softened like chalk and it may be dug into easily with suitable sharp instruments. If the quantity of diluted, weakened or buffered acid is frequently renewed, this partial decalcification may extend deep into, or in time all the way through, the enamel. In this way I have partially decalcified the entire enamel cap, which could then be shelled off in large pieces from the underlying dentin. If, on the other hand, all the enamel is covered with wax, except for a limited area, then only the uncovered area which is exposed to the acid is partially decalcified. This corresponds to the natural early stage caries process.

If we use organic acids such as lactic (which is supposed to be the principal one involved in the natural caries process) the same phenomena occur except that, in general, the action is much slower. When such weak acid solutions are used the enamel cuticle remains intact and in place. Thus weak acids pass through the cuticle and the products of their action on enamel pass back out through this very delicate osmotic membrane without destroying or impairing it.

In this way the early stage* lesion in natural enamel caries is produced. Acids produced at the surface of the bacterial film, and perhaps also deeper within it, pass along through this sponge or wick-like material, which we have seen is composed of countless millions of long filamentous type organisms, down to the cuticle and thence through it into the enamel.

This is a long slow microscopic and microchemical process resulting from the often repeated slow action of delicate amounts of weak acids produced on or

* (For convenience the term early stage may be applied to caries lesions before there is any break of the surface or cavity, and advanced stage where a cavity, of whatever size, has developed.)

within the bacterial film at the particular location. Time is required for the passage of such weak acid through the bacterial film, (Fig. 9) at the same time interchanging with, and being diluted by fluids of different reaction and composition before it reaches the surface of the tooth which is covered by enamel cuticle. Time is also required for osmotic passage of acid through the cuticle against substances of different reaction and composition resulting from the action of previously introduced acids upon the enamel.

Fig. 9. Ground section (unstained), very early stage caries. Note variation in depth to which process has advanced in different enamel rods. Also note pad of bacterial film, Ba, at surface.

Still more time must be required for passage of the acid into the enamel, especially as the partial decalcification extends deeper and deeper. Not only must the acid pass through the already partially decalcified enamel to the depth of the process but there is a great tendency for this process to follow the course of individual enamel prisms. In suitable preparations under high enough magnification, we usually find the acid action extending along one prism farther than another (Fig. 10) in the immediate vicinity. This means that acid arriving at already partially decalcified enamel passes along the course of the individual prisms until it reaches the farthest point to which previous acid action has extended. Along this course of the prism the minute acid must interchange with products of previous acid action and finally reach the point of activity in effective quantity and strength. Again this is a very slow process. Thus the chalky enamel caries lesion represents the cumulative effect, over considerable periods of time, of minute amounts of weak acids produced, at various times, by bacterial action upon carbohydrate food material at the particular location.

Fig. 10: Ground section through farther advanced chalky enamel. Note variation in depth to which process has advanced in different rods and in intensity at different locations.

Pertinent facts relative to the caries process in dentin.—If the partial decalci-

fication of enamel (early stage caries lesion) continues to progress, it finally reaches the dentin. Minute quantities of acids produced at the surface now slowly pass through the chalky enamel, especially along the cracks (lamella) and channels that may exist there, thus reaching the dentino-enamel junction and on into the dentin. The caries process in dentin differs substantially from the process in enamel. This results largely from the different composition and structure of dentin.

Dentin consists of about 30 per cent organic matter and water, and 70 per cent inorganic material.[26] Acids remove the inorganic material leaving the cartilage-like dentinal matrix material which is now softer and can easily be cut or torn apart with suitable instruments.

During the dentinal caries activity decalcification advances deeper and deeper into the dentin, and the decalcified dentin at the surface (bottom and walls) of the cavity slowly disintegrates, thus increasing the depth and size of the cavity. Some of the bacteria of different kinds within the cavity grow and advance into the dentin by way of the open-ended dentinal tubules, towards the pulp chamber.

Near the surface of the cavity wall all the tubules are packed with bacteria. (Fig. 11). The pressure of the growing and enlarging mass of multiplying bacteria within the tubules in the decalcified dentin expands the tubules, compresses the decalcified softened matrix material and tends to split and break it up. Dislodged particles of the decalcified dentin are continuously being shed off from the disintegrating surface, thus increasing the depth and size of the cavity. (Fig. 5). Much deeper in only part of the tubules contain bacteria, many others at the same level do not. (Fig. 12). This means that the forward advance of bacteria into the dentin proceeds down separate tubules in-

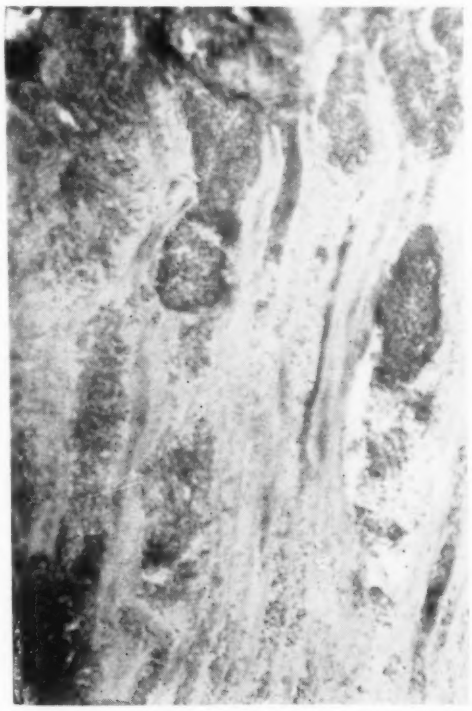

Fig. 11: Section through bottom wall (at top) of cavity in dentin. Note bacterial masses in softened dentin and some extending into dentinal tubules from surface.

Fig. 12: Bacteria advancing pulpward down separate tubules. Others at this same level not yet invaded.

dependently of other nearby tubules.

The dentinal tubules extend through the dentin from the pulp chamber to the dentino-enamel junction. Their size near the pulp is several times that near the outer surface.[26] They contain long protoplasmic projections (Tomes fibers)[27] from the odontoblasts. In normal vital dentin a nutritional fluid, dental lymph,[28-30] circulates in the dentin, principally through the dentinal tubules and their many anastomosing branches.

Irritation from severe wear, erosion and caries stimulates the formation of secondary dentin (irregular dentin, Orban[26]) at corresponding areas on the pulpal wall. This new-formed secondary dentin effectively closes off the tubules of the involved regular dentin. This is now spoken of as a dead tract. Dental lymph cannot pass through the now closed tubules of the area and bacteria cannot advance toward the pulp, as they can in open tubules.

No doubt laying down of this wall of secondary dentin which closes the tubules and shuts off the passage of dental lymph into them, is nature's most important protection against advancement of bacteria into the tubules, and ultimately infection of the pulp. Bodecker[29] has called these changes "protective metamorphosis."

Cervical Caries.—The cervical caries lesion begins at the cemento-enamel junction, and only after the gum has receded enough to expose the area. For unknown reason some people seem to be especially susceptible to this type of lesion. This is probably related to varying relationships in the area where enamel and cementum join.[26-31] In some instances their edges do not quite meet, thus leaving an area of dentin covered only by soft tissue. After this is removed there is left an area of exposed dentin with open-ended tubules, which does not have the usual protective covering of either cementum or enamel. They are now open to invasion by bacteria.

In other instances the cemento-enamel junction presents a V-shaped groove of considerable size which, after the soft tissue is removed, offers a favorable situation for accumulation of filamentous type bacteria, as occurs elsewhere in grooves and depressions.

Whatever the special conditions may be that initiate the cervical caries lesion, it advances into the dentin in much the same way as do lesions originating in the enamel after they reach dentin.

Inactive Caries (Arrested Caries).—Caries lesions usually begin at the vulnerable locations during the first two or three years following eruption of the particular tooth. If the local etiological conditions which initiated the lesion continue, the disease process continues to advance. In time a cavity (advanced stage lesion) develops large enough to be recognized by the patient or by the dentist. The preceding early stage chalky enamel lesion has existed and progressed unrecognized for months previously.

If at any time, the local conditions which cause the lesion and promote its advance are prevented or altered sufficiently, the progress of the disease process slows down or stops entirely. This may occur at any stage of the lesion from the earliest (microscopic) chalky enamel stage to the advanced cavity stage. Obviously the earlier this occurs the less damage will have been done.

A large part of all proximal caries lesions stop advancing—become inactive—before cavities are formed. Often small cavities in this region also cease to advance. This slowing down or cessation of proximal caries activity coincides with the incidence and increasing activity of periodontoclasia in the same region. Bibby[32] has recently reviewed the literature relative to the apparent antagonism between caries and periodontol disease. The available information cited tends to support some such antagonism.

Gingivitis is characterized by a continuous flow of minute quantities of inflammatory exudate which consists of serum containing pus cells and some blood cells. Constant presence of such exudate in the

environment of the caries lesion between the teeth tends to neutralize and counteract the effect of minute quantities of weak acids in the area. It is possible also that it may influence the local bacterial flora and promote the growth of proteolytic bacteria in the environment, thereby lessening acid production by acidogenic bacteria. On the other hand, periodontoclasia has little or no influence upon occlusal caries. Inflammatory exudate does not reach the area in effective amounts and concentration.

Early stage caries—chalky enamel—always appears pure white throughout the life of the tooth, as long as the cuticle remains intact. Usual stains of the mouth do not pass through normal cuticle. On the other hand, chalky enamel from which the surface has been destroyed becomes stained—usually varying shades of brown. Stains are also carried into chalky enamel through cracks (lamella) and other channels that may exist there. The staining of such lesions tends to increase, although they may remain inactive, throughout the life of the tooth. Usually chalky enamel lesions which had advanced sufficiently to be stained because of loss of cuticle or penetration of lamella or small cavities remain white around the borders in areas in which the cuticle is still intact.

Occlusal caries lesions start at the entrance and sidewalls of occlusal pits and fissures of molars and bicuspids where the depressions favor accumulation of bacterial film, retention of food material, and production of acids. The partial decalcification—chalky enamel stage—progresses and finally cavities develop. There is a tendency for the decay process to extend down any channels and after it reaches the dentin, to spread and undermine the enamel along the dentino-enamel junction.

Extensively undermined enamel may crack off leaving the decaying dentin widely exposed. Bacterial film and food material in such wide open occlusal cavities may be disturbed or rubbed off several times every day. Occasionally dentinal decay in wide open cavities ceases to advance and the dentin becomes very hard, similarly to the way in which dentin exposed by severe wear becomes almost as hard as enamel. This is an example of what often happens when such a wide open cavity is frequently cleaned out by whatever means.

Sometimes radical changes in diet habits or in oral hygiene habits and methods at any time, may slow down or stop the activity of early stage occlusal caries lesions. However, in most instances, no such circumstance arises, especially during the age period (up to 12 or 15 years of age) of greatest activity of such lesions.

Periodontoclasia-Definition. — Various terms have been used to indicate the disease process encompassed by periodontoclasia. Such terms as periodontal disease, periodontosis, periodontitis, pyorrhea, pyorrhea alveolaris, alveolodental pyorrhoea, Riggs disease, gingivitis have been applied to the disease in general or to some particular phase or prominent clinical condition. The disease is characterized by inflammation and suppuration of the epithelial tissue within the gingival crevice, and by long continued progressive chronic inflammation in the parodontal tissues resulting in destruction and resorption of periodontal fibers and alveolar bone.

Periodontoclasia is caused by foreign material, consisting of bacterial film and/or concretions (mostly calculus) upon the surface of the tooth at the entrance to, and within, the gingival crevice. Prevention of the disease and prevention of further progress of existing lesions can be accomplished only by preventing or minimizing these local etiological conditions—conditions without which lesions do not originate or advance.

Early stage of the disease.—All periodontoclasia lesions begin at the gingival margin and are, at first, only microscopic in extent, therefore not recognized.

It has been pointed out [1, 10, 12, 14] and emphasized above, that bacterial film (plaque) on protected areas on the surface of the tooth consists of a pad of filamentous types of micro-organism, one end attached

to the tooth and the other extending outward towards the surface of the pad. At the surface of the pad there may be found more or less of any and all of the many other kinds of bacteria in the mouth.

At the entrance to the gingival crevice this bacterial material tends to grow or advance into the crevice and to impinge upon the epithelial surface there. The deeper part of the bacterial material continuously present upon the tooth tends to harden, calcify, and form rough concretions there. (Fig. 13). Normally, the marginal gingival tissue rests upon the soft smooth non-irritating enamel cuticle. Hard, rough material superimposed upon the cuticle acts as a mechanical irritant like a foreign body.

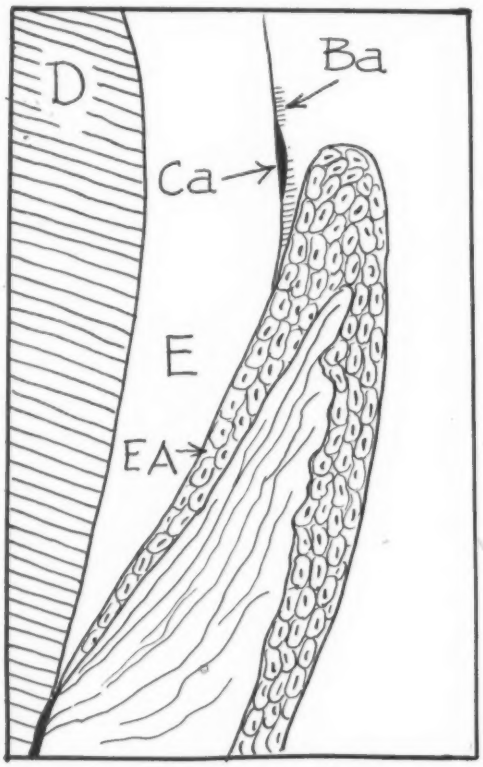

Fig. 13: Drawing to indicate foreign material at entrance to gingival crevice in early stage periodontoclasia. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, EA-epithelial attachment.

Soon the gingival margin is irritated by this foreign (and perhaps toxic) material against which it rests. Inflammation of the epithelial tissue ensues, characterized by migration of polymorphonuclear cells through the affected tissue (Fig. 14) to the point of irritation, where they tend to accumulate as pus cells.

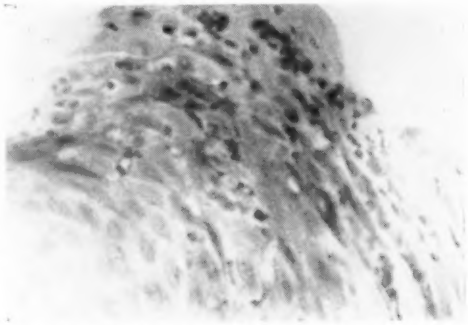

Fig. 14: High magnification of section of marginal gingiva showing polymorphonuclears passing through the epithelial tissue at right which was in contact with irritating material on tooth. Good oral epithelium at left.

At first the amount of inflammatory exudate, which consists of pus cells and serous fluid, is not sufficient to be recognized by the unaided eye. However, material properly collected from the location always contains pus cells, upon microscopic examination; and, sections always show polymorphonuclear cells passing through the epithelial tissue opposite the irritating foreign material on the tooth against which it rested.

As the inflammation continues the bacterial film on the tooth tends to extend deeper and deeper into the gingival crevice, (Figs. 15, 16) the amount of the associated calculus on the tooth within the crevice increases, and there is increase in the amount of pus produced. The inflamed slightly congested and edematous condition of the marginal gingiva may now be recognized upon careful examination. It bleeds easily upon slight pressure or manipulation. The condition may now be noticed by the individual and by the dentist. Usually the importance and significance of gingivitis, as the early stage of the periodontoclasia lesion is not recognized. However it is, in fact, the begin-

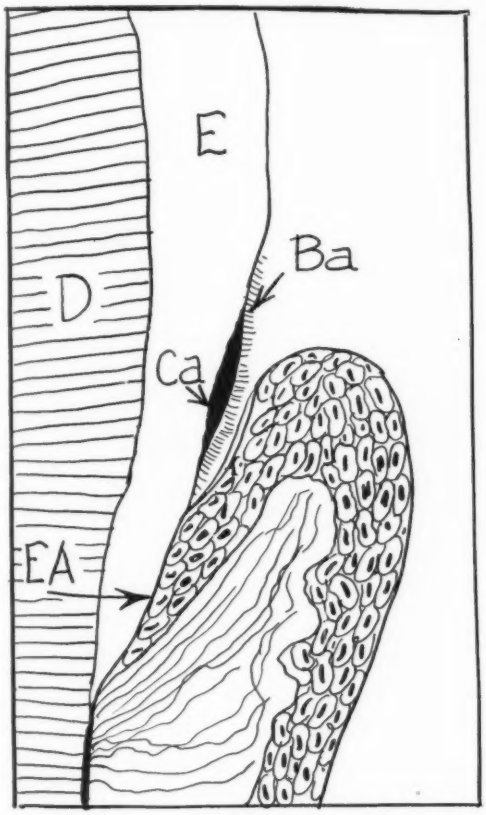

Fig. 15: Drawing to indicate foreign material within the gingival crevice in an advancing periodontoclasia lesion. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, EA-epithelial attachment.

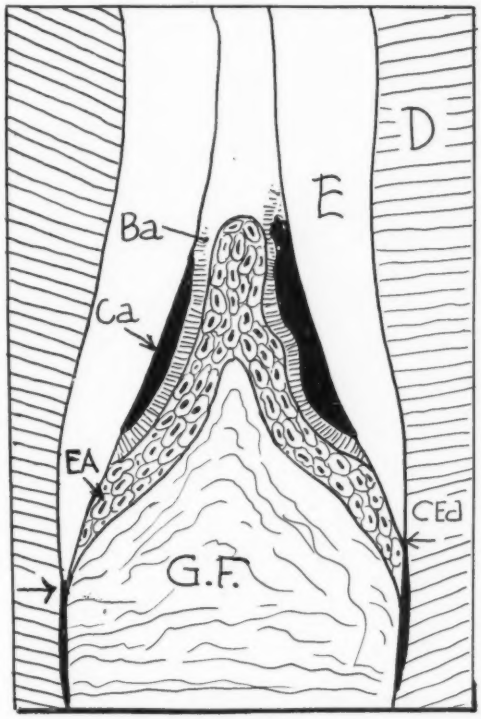

Fig. 16: Drawing to indicate foreign material within the gingival crevices in advancing interdental periodontoclasia lesions. Ca-calculus, Ba-bacterial film, E-enamel, D-dentin, CEJ-cemento-enamel junction, GF-gingival fibres.

ning of a progressive pathological process which never ends spontaneously, until the tooth is finally lost. The origination, activity, and rate of advancement of early stage lesions, and also, of the more advanced stage lesions, are determined largely by the effectivenes of the personal oral hygiene habits of the individual. These vary greatly with different individuals, at different times, and as applied to different tooth areas.

Nature of the subgingival bacterial film and calculus.—Once the foreign material (bacterial film and calculus) has advanced into the crevice and inflammation is set up there, more or less inflammatory exudate is continuously present in the crevice thereafter. This exudate consists of diluted serum (lymph) containing pus and blood cells. It provides a favorable environment for the establishment and growth of types of micro-organisms which are favored by partial anaerobiosis and by the serum-rich substrate. Such filamentous type organisms as actinomycetes and certain leptotrichia are encouraged.

Subgingival calculus consists of calcified bacterial material.[33, 34]. The inorganic portion is derived from the inflammatory exudate (serum). The bacterial portion consists largely of filamentous types of microorganisms, especially actinomyces.[35]

A pad or film of filamentous type organisms is always present on the surface of the tooth, and on the surface of any calculus, within the periodontoclasia pocket. (Fig. 17). This constantly present

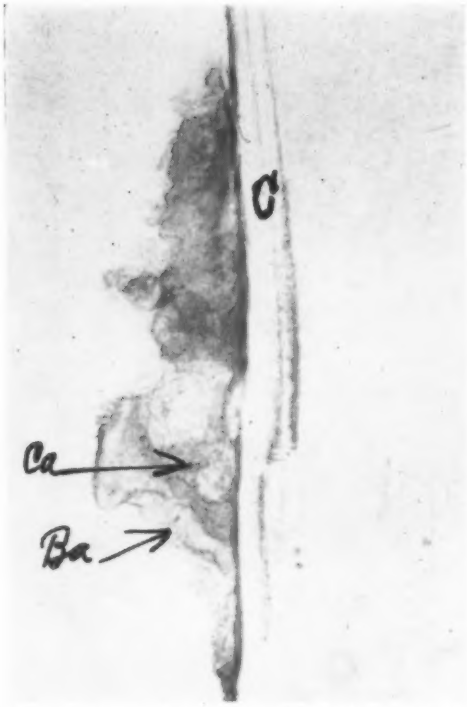

Fig. 17: Section through foreign material, which was located on the tooth within the periodontoclasia pocket. C-cementum, Ca-calculus covered by heavy lighter-colored bacterial film Ba.

bacterial film is composed of long filaments with one end attached to the tooth or to the calculus, (Fig. 18) and extending outward in palisade form to the surface of the pad which is in contact with the inflamed crevicular epithelial surface. At the surface of the pad there are the growing ends and fruiting heads of the particular organisms composing the film pack.

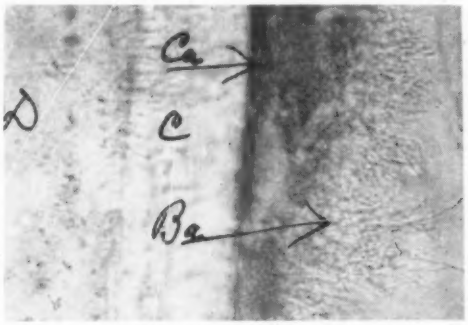

Fig. 18: Section through side of tooth which was located within the periodontoclasia lesion. C-cementum, D-dentin, Ca-calculus (darker) covered by filamentous type bacteria Ba, right. Note dark previously undescribed pathological [89] granules in dentin.

One of the important thread form organisms usually found in the pyorrhea pocket was first described by Beust [36] and named by him *Leptothrix falciformis*. He later [37] called attention to this organism in material from about the teeth, and pointed to the association of it with spirochetes.

I [38] have observed that the habitat of *Endameba buccalis* is the outer surface of the filamentous bacterial film on the tooth within the periodontoclasia lesion. "There they are protected and live, grow and multiply among the strands and fruiting heads of leptotrichia, principally *L. falciformis*." This observation conforms with the observations of others [39-42] as to the distribution of this parasite in the periodontoclasia lesion. Kofoid [39] confirmed especially the observation [43] that ameba are most numerous at the very bottom of the pyorrhea pocket. This ameba cannot be the specific cause of periodontoclasia, as sometimes has been erroneously supposed, for the reason that it is found in the lesions of not more than about 50 per cent of adults under 30 years of age. This does not necessarily mean that the parasite may not be harmful whenever it is present.

The fact has been shown [44] that the film pack is composed of filamentous types of micro-organisms, largely leptotrichia, attached to the surface of the tooth or to the calculus within the periodontoclasia lesion and that there is a tendency of the fruiting heads of *L. falciformis* to extend into the deepest part of the lesion. This conforms with observations of Box [45] in this regard.

It has been observed [44] also that the film pad of *L. falciformis* in the deeper part of the periodontoclasia lesion, and the conditions there, are especially favorable for the growth of spirochetes. These

are found in greatest abundance upon and among the fruiting heads of the leptotrichia at the very bottom of the pocket, and therefore overlying the zone of disintegrating epithelial attachment cuticle (zdeac).[46] This zdeac serves as a useful landmark which accurately indicates, on extracted tooth specimens, the location of the outer border of the epithelial attachment and the bottom of the periodontoclasia lesion[47] at any place around the tooth when it was in situ. The great abundance and constant presence of spirochetes at this location, which is where the lesion is advancing on the surface of the tooth—the location where the outer border of the epithelial atachment is receding apexward—suggests the possibility that spirochetes have some etiological significance in the advancement of the lesion.

While the flora and fauna tend to be limited to fewer varieties in the deeper part of the pocket, the entrance to the crevice and the part just within, are constantly exposed to infection and reinfection by any and all of the many species in the mouth. Material collected from this location always contains more or less of such mouth organisms. It is interesting to mention also that this location is where fusiform organisms are most abundant in mouths in which they and associated spirochetal organisms predominate.

Apexward advancement of the subgingival bacterial film and calculus.—The subgingival bacterial film and/or calculus tend to continuously advance apexward at various rates at different locations on a given tooth and on different teeth in the same mouth. As a result of the inflammation, suppuration, and destruction of the parodontal tissues which this foreign material on the tooth within the crevice causes, the location of the epithelial attachment moves apexward,[47, 61, 66] thus deepening the pocket and leaving more and more of the tooth without its normal soft tissue covering. Such bared tooth surface is always entirely covered with bacterial film right down to the outer border of the epithelial attachment.[44] Calculus formation follows along not far behind.[48] However, there is always a narrow band or space, of variable width, between the inner (or deeper) border of the calculus and the outer border of the epithelial attachment.

The epithelial atachment consists of a thin band or cuff of epithelial cells surrounding the tooth. The width of this cuff varies at different locations. It is widest when it is located on the enamel and becomes narrower as it moves apexward[46, 47]. Waerhaug[49] has presented strong evidence that what has been called the epithelial attachment is, in fact, not organically attached to the surface of the tooth and that a delicate instrument can be passed, with little resistance, into the gingival crevice all the way to the cemento-enamel junction. Baume,[50] through phase contrast microscopy, finds evidence that enamel epithelial cells are very delicately

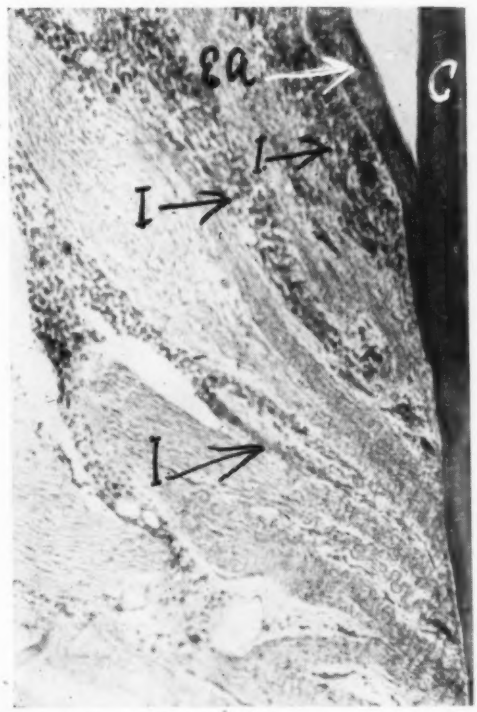

Fig. 19: Epithelial attachment EA, advances apexward over cementum, C, from which periodontal fibers have been destroyed. Note inflammatory cell infiltration I among fibers. (Attachment pulled away from cementum at top.)

attached to the tooth surface by tonofibrils, and that similar fibrils hold together the cells of enamel epithelium, and also those of the adjacent oral epithelium.

The inner border of the epithelial attachment grows apexward to cover over cementum from which periodontal fibers have been destroyed and removed (Fig. 19). As more fibers are destroyed, the epithelial attachment advances farther. Destruction and resorption of the periodontal fibers which make possible the advancement apexward of the epithelial attachment, and which gives to the disease its name—periodontoclasia—results from chronic inflammation extending into the periodontal tissue among and between the periodontal fibers for considerable distances from the bottom of the pocket. (Fig. 20). This inflammatory process is characterized by round cell and plasma cell infiltration, and by accumulation of these inflammatory cells between the bundles of periodontal fibers. (Figs. 21, 22, 23, 24). As the process continues the involved fibers are broken down and finally resorbed.

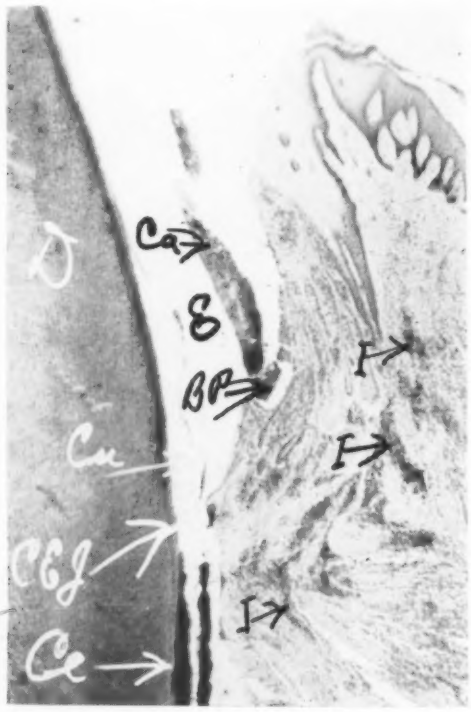

Fig. 20: Section of typical active periodontoclasia lesion (slightly pulled apart). Note especially, location of calculus, Ca, in relation to inflamed gingival tissue. Note also how far round cell infiltration, I, where periodontal fibers are being destroyed, extends from bottom of pocket, BP. E-enamel space, Cu-ribbon of enamel cuticle, D-dentin, Ce-cementum, CEJ-cemento-enamel junction.

The important fact that foreign material (calculus and bacterial film) is always present on the tooth within the inflamed gingival crevice (the periodontoclasia lesion) is well shown in numerous illustrations to be found throughout the literature relative to this disease, or in some instances presented for other purposes. Most pictures show what is usually labelled calculus on the tooth within the pocket. The fact that the calculus and the

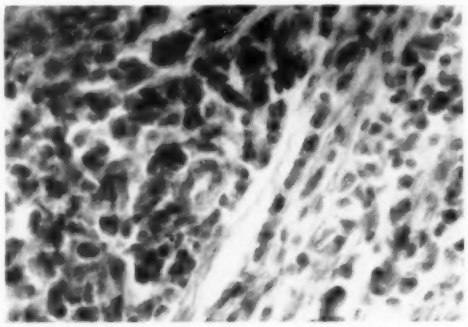

Fig. 21: Heavy round cell and plasma cell infiltration in periodontal tissue resulting in destruction and removal of periodontal fibers. Only a few fibers remain.

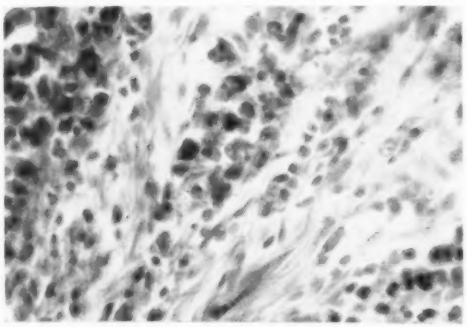

Fig. 22: Round cell and plasma cell infiltration among and between periodontal fibers. Destruction of fibers not quite as advanced as in Fig. 21. More fibers intact.

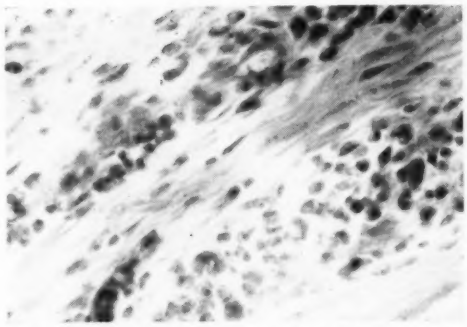

Fig. 23: Inflammatory cell infiltration less than in Fig. 22. Good periodontal fibers intact.

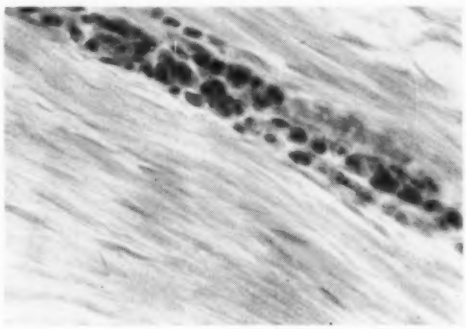

Fig. 24: Inflammatory cells accumulated in a streak between good intact fibers. Condition often found farthest from the bottom of pocket.

part of the tooth between the inner border of the calculus and the outer border of the epithelial atachment are covered by heavy bacterial film,[44, 48] is usually overlooked. Following are listed a few of such publications and the particular illustrations which show this important feature of the disease, upon which effective personal oral hygiene must be based: [51](Figs. 1 2, 3, 4), [52] (Fig. 6), [53] (Fig. 8) [54] (Figs. 20, 23, 29), [55](Figs. 2, 7, 8), [56](Figs. 1, 5, 6), [57](Figs. 3, 12, 14, 20, 25, 26, 27, 29, 30), [49](Figs. 17, 18, 20, 39), [58](Fig. 10), [59] (Figs. 24, 26, 31), [60] (Figs. 16 (2), 13 (3), 24 (7)), [16](Figs. 278, 287, 288, 289, 293, 305), [62](Figs. 3, 4), [63](Figs. 3, 5), [64](Fig. 190), [26](Figs. 138, 218, 222), [65](Figs. 59, 64), [18](Fig. 104), [67](Figs. 491, 713, 733, 734, 735, 738, 767B), [68](Figs. 261, 264, 268), [69](Fig. 15), [70](Figs. 6, 8).

The lesion, at first microscopic and later macroscopic, extends around the tooth, but is usually deeper and more active in the interproximal crevices which are deeper and where accumulations of foreign material are less subject to removal by functional friction or customary methods of oral hygiene. The tooth surrounded by concretion and bacterial material adhering to it within the gingival crevice (lesion) is, in effect, a foreign body, constantly subject to infection and reinfection with the many different kinds of bacteria in the mouth.

This foreign body effect was recognized and its importance emphasized more than seventy-five years ago by John W. Riggs.[71] More than sixty-five years ago J. N. Farrar [52] urged the necessity of removing this subgingival foreign material and laid special emphasis upon keeping the area clean. He said "the disease vanishes when the pockets are carefully and thoroughly cleaned, and *kept clean.*" To this day, removal of this material from the tooth, which these pioneers advocated at that time, is still essential for successful treatment of the disease. Not only is it essential, but it is perfectly logical, that the local cause of the disease should be removed and its reformation prevented. Prevention of its reformation is accomplished by the method of personal oral hygiene herein presented.

(To be continued in March issue)

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COMPULSORY EXTENSION OF SOCIAL SECURITY COVERAGE TO PRIVATE PHYSICIANS

At present, physicians are exempt from the operation of the so-called Social Security law, which is the popular name for the Old Age and Survivors Insurance phase of the Federal Welfare program. In a recent message to Congress, President Eisenhower recommended the compulsory extension of social security to three and a half million self employed persons, including private physicians. Congress will soon act upon this legislation, and it behooves each member of organized medicine to consider thoroughly the implications of such laws and to be able to talk to his congressman on a factual basis in regard to their passage.

The House of Delegates of the American Medical Association, in 1949, and again, in 1953, expressed strong opposition to the extension of compulsory coverage of physicians under Title II of the Social Security Act. At the same time, it expressed approval and support of the Jenkins-Keogh Bills (HR 10 and HR 11) which are now before the Congress. These bills are designed to provide for the development of a voluntary pension program which is equitable, free from compulsion, and satisfies the retirement needs of physicians on a voluntary basis.

If social security were extended to physicians, it would levy a 3 per cent tax on the first $3600, and later, probably, the first $4800 of income. This would provide $85 a month after age 65, and an additional $45.50 for the wife after she reaches 65, while he is living, and $63.80 when she would become a widow. However, if a physician earned more than $900 a year after 65 he would forfeit his social security benefits until after age 75. From the physician's point of view such a bill would serve the improvident and would afford considerable financial advantage to those who enter it at an advanced age. Dr. Dickinson states that a self employed person at age 62, brought under Old Age Insurance in 1952, by paying as little as $121.50 in OASI taxes, could have a pension of $80 a month for himself, $40 a month for his wife and $60 a month for his widow after his death. On the other hand, an insurance company would want about $23,000 at age 65 for such a contract.

Against this type of legislation, are many facts which would make it an unjust tax on physicians as a whole. The most important reason against compulsory Social Security for physicians is the fact that it is another move in the direction of regimentation and the welfare state. The long-time result would be big pensions

and bigger government. The prohibition against earning more than $900 while on Social Security would make it inacceptable to most physicians. The majority of them do not retire, and if they did, they could not live on $85 a month. The fact that he is allowed to receive income from his investments is of no value in the argument, because if such income is going to be sustaining, there is no need for his enforced inclusion anyway.

The proposed payments under the anticipated change in the Social Security law would not bring a pension paid for in advance, but, on the contrary, would simply be a dole provided from the tax payments of younger participants and other taxpayers. It is calculated in the report of the Curtis Subcommittee on Social Security of the House Committee on Ways and Means that beneficiaries now on the OASI rolls have prepaid with their employers only about one twenty-fourth of the cost of the benefit they are receiving. Another portion of the money is coming from the payments of the younger taxpayers and from the general taxpaying public.

As physicians we are cautious and our experience makes us into conservatives. As such in recent years when there has been such an avalanche of social legislation designed to regiment the medical profession and establish state medicine, we have frequently been put in the attitude of opposing these unworthy projects. We have been criticized for being against everything, particularly, this form of so-called progress. In opposing the extension of compulsory inclusion in the Social Security Act we are able to endorse and support a constructive piece of legislation designed to do the same thing that the Social Security Act ostensibly would do, and do it much better as an expression of free enterprise. The Jenkins-Keogh Bills, which were mentioned above, and which have previously been discussed in these columns, would accomplish this. These Bills do not amend the Social Security Act. They do amend Section 165-A of the 1942 Federal Internal Revenue Code. These bills would allow tax deferment for individual retirement funds. They would benefit 11,000,000 self employed. They also would benefit 30,000,000 pensionless employed, whose employers have no pension plans. Thus, it is seen that two thirds of the 61,000,000 gainfully employed persons are pensionless. While these self employed and many of these pensionless employed lack the employee fringe benefits which characterize our era the Jenkins-Keogh Bills would supply them. These pensions drawn from the individual retirement funds would range from 16 to 32 per cent of average annual earnings compared to the benefits from the tax exempt pension plans of the twenty largest employers in the United States, which range from 34.6 to 53.2 per cent of annual earnings.

Along with the American Medical Association, the American Dental Association, the American Farm Bureau Federation, and the American Bar Association, have approved these tax deferment individual retirement fund proposals. Organized medicine regards these bills as the answer to compulsory pensions. It recognizes that pressure for compulsory health insurance follows in the wake of compulsory pensions. The socialist bureaucrats have failed to establish socialized medicine by a frontal attack. They now seek to reach their objective by adding health benefits to the Social Security system, on the one hand, and federal subsidies to the voluntary health insurance on the other.

The problems that Medicine faces along these lines are clearly delineated in recent publications of the American Medical Association.[1,2] These plans will require consideration by physicians during the years

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1. Blasingame, F. J. L., and Dickinson, Frank G.: A physician and an economist look at old-age and survivors insurance, J.A.M.A. 153:921, (November 7) 1953.

2. Dickinson, Frank G: Prepaid pensions for all-including physicians, J.A.M.A. 154:417, (Jan. 30) 1954.

ahead and all should familiarize themselves with the substance of these two discussions.

The Jenkins-Keogh Bills would be very helpful. The operation of compulsory Social Security would ultimately operate for medical regimentation. In substance, this tax will provide very little for the average physician, and is just another plan among many which converge to make the productive half of the population carry the other half until they both drop.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

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MEETING OF STATE CHAIRMEN AMERICAN MEDICAL EDUCATION FOUNDATION

Following is report submitted by Dr. Edgar Hull, member of the Committee on American Medical Education Foundation

of the State Society, who attended a meeting of state chairmen held in Chicago, Sunday, January 24, 1954.

REPORT

Dr. Turner spoke about the purposes and aspirations of the AMEF. He spoke of the great increase in the cost of medical education in the past two decades, both to the school and to the student. He emphasized especially the importance of help to the students, who have to pay three times as much for their medical education as they did two decades ago. He mentioned the importance of the schools and the students being helped without their being forced to seek federal aid with its inevitable federal control. He stated that there were two sources from which such aid could be obtained: (1) from physicians, and (2) from the lay public. AMEF is dedicated to solicit funds from physicians, the National Fund for Medical Education to solicit funds from the lay public, especially from industry; and that the two funds were being pooled, divided among the schools, and given to them as unrestricted gifts.

It occurred to me while Dr. Turner was speaking that it might be advantageous if the schools were to use some of the funds which they receive from the Foundations in order to establish loan funds or scholarships for their students.

Dr. Turner and others emphasized the fact that the AMEF has no administrative costs, all of which are borne by the AMA, so that every dollar which a doctor contributes to AMEF goes to the medical schools.

Mr. Ade, Director of Fund-Raising for the National Fund for Medical Education, stated that the fund is now in the process of trying to sell industry on their stake in medical education, and trying to get industrialists to give to the central fund to help all medical schools rather than to contribute to individual medical schools. He said that part of their task has been to educate business men as regards the stake of industry in medical education, and to point out that medicine is one of the last bulwarks in resisting governmental control. Business men are being told that if industry does not contribute to the needs of the medical schools, government must and will take over. He pointed out, however, that industry will not support medical eduation unless the doctors themselves support it as well. He was pleased, however, that during the past year slightly more than half of the total amount distributed to medical schools came through direct and indirect contributions of physicians. He stated that the goal of the combined foundations is ten million dollars per year, two million of which is to be raised by AMEF and the rest by the National Fund.

Mrs. Gastineau spoke of the activities of the Woman's Auxiliary of the AMA and of the State Auxiliaries in helping the campaign of AMEF.

Mr. Pettis, Associate Director of the California Medical Association, spoke of the Audio-Digest Foundation, which is now a subsidiary of the California Medical Association. This foundation is preparing and selling tape recordings which digest medical literature for general practitioners and specialists, and which is also recording for distribution lectures and round-table discussions by leading authorities. Audio-Digest was originally started as a private corporation which was growing rapidly before the California Medical Association took it over. All profits of the Audio-Digest Foundation are to be given to AMEF, and it is believed that at its present rate of growth this should amount to nearly a million dollars a year within the next few years.

Mr. Pettis stated that exhibits explaining Audio-Digest are now available for State Medical meetings. It would be my recommendation that their exhibit be shown at the State Meeting this year.

Mr. Jones, Executive Secretary of AMEF, spoke of the increase in contributions by doctors in 1953 over 1952, but recalled the fact that the eventual goal of two million dollars per year has only been half realized. It seemed to me, however,

that actually the goal has been only one-fourth realized, since of the little more than a million dollars raised by the Foundation in 1953, a half million was a direct grant from the AMA.

Mr. Jones emphasized again the fact that grants to schools are entirely unrestricted, that the Foundation has no administrative costs; he also mentioned the fact that when doctors contribute to AMEF they may specify the schools to which they want their contributions to go. He mentioned that two states, Illinois and Utah, had increased their dues by $20.00 annually, the extra $20 to go to the AMEF, and stated that in Illinois, which has led all other states in contributions to AMEF, the increase in dues did not result in a decrease in individual contributions to the Foundation. As I understood it, both in Illinois and Utah physicians who believe that the increase of $20 will work a hardship upon them are excused from paying this extra amount in dues. He stated also that New York has a voluntary plan in operation whereby each doctor, at the time he is sent the statement for his dues, is requested to include an additional $10 to go to AMEF. He suggested an eventual goal of $25 contributions from each member of every State Society. He believes that the principal function of the Woman's Auxiliary as regards AMEF is to sell their husbands on the idea of contributing.

At the afternoon seminar a number of questions were asked and answered. The main points which seemed to me to be of importance were as follows:

1. How the funds are divided among the medical schools. At present there are two bases for such distribution of funds: (a) a uniform sum, which goes to all medical schools regardless of their size; (b) a pro rata sum based upon number of students in the school. It is planned in the future, as the size of the fund increases, to provide an additional type of grant based upon the special needs of certain schools. It was noted on sheets which give the amounts paid to each medical school that the amount to each is proportional to the number of students, and that two-year schools get half as much as the four-year schools.

2. Mr. Steinwell, from Maryland, who apparently is connected with Johns Hopkins University, stated that there had been conflict between the AMEF campaign and Hopkins' own annual campaign to raise funds from their alumni. He stated that they preferred for their alumni to contribute directly to Hopkins rather than to AMEF, and he felt certain other endowed schools would have the same opinion. There was a lot of discussion about this, and it was pointed out by Mr. Ade that the prime purpose of AMEF and the National Fund was to get persons to contribute to all medical schools, so that the small as well as the large ones, and those with large active alumni associations as well as those with smaller groups of alumni might be helped. No action in this regard was taken at this meeting, but Mr. Jones stated that they had conferences in mind to discuss this problem.

3. Near the close of the meeting Mr. Ade, of the National Fund, stated that he is anxious to set up local committees in various cities in order to establish machinery for the solicitation of men in business and industry for contributions to the National Fund. It is my recommendation that the State Society do whatever it can to increase the number of physicians who make contributions to AMEF, but I have no specific recommendation as to how this should be done. It is noted on the State list of contributions that in 1953 there were only 39 physicians in Louisiana who contributed, and that their total contribution was only a little over a thousand dollars. This places Louisiana near the bottom of the list, and I believe actually at the bottom in proportion to its population.

Edgar Hull, M. D., Member
Committee on American Medical Education Foundation

The above report by Dr. Hull should stimulate the members of our Society who

have aided this fund in the past to contribute more liberally. It should also be an incentive to the other members who have not given any financial support to this most worth-while undertaking, sponsored by these national groups, to contribute, thereby aiding our two medical schools in continuing to give our medical students the splendid medical training offered in the past.

As an alumnus of either school, you can earmark your contribution and designate to which school you desire your contribution sent.

This amount so earmarked for the school of your choice will, in no way, reduce the amount to be received by the respective schools from the grand total collected by these two national organizations. It does not matter whether you send your contribution direct to the American Medical Education Foundation or to the school of your choice, it will go to the school for which the gift is earmarked and intended. You will be given credit for your contribution, in either case, in their annual reports.

These funds, so earmarked will not be included in the General Fund, and your school will still receive, in addition, the proportional amount along with other schools from the total funds allotted for all medical schools.

Most assuredly we as members of our State Society, should be ashamed to let our state be at the bottom of the list as it is stated to be in Dr. Hull's report.

Let us all take a pledge to make a better showing for 1954.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

EENT ANNUAL MEETING

The annual meeting of the Eye, Ear, Nose and Throat Hospital Alumni Association will be held in New Orleans at the Hospital April 5, 6, and 7, 1954. Samuel Fomon, M. D., of New York City will deliver the Clyde Lynch Memorial Lecture and participate in operative demonstrations. All Alumni and other interested E.E.N.T. men are invited to attend.

FIFTH DISTRICT MEDICAL SOCIETY

At a meeting of the Fifth District Medical Society, held with the Ouachita Parish Medical Society in Monroe on December 10, the following doctors were elected to serve as officers for 1954:

Dr. Henry E. Guerriero, Monroe—President.

Dr. W. J. Burchell Liles, Monroe—Secretary-Treasurer.

Dr. F. McC. Terrel, Lake Providence—Delegate.

Dr. Mortimer Raphael, Monroe—Alternate.

Dr. Jack G. Kerr and Dr. LeRoy J. Kleinsasser, both of Dallas, presented scientific papers on "Infectious Ano-rectal Disease" and "Treatment of Acute Vascular Disease", respectively, and Judge Vincent Mouser, of Columbia addressed the group after dinner was served to members and their wives.

THE SOUTHEASTERN SURGICAL CONGRESS

The Twenty-second Annual Assembly of the Southeastern Surgical Congress will be held in Birmingham March 8-11, 1954, with headquarters at the Dinkler-Tutwiler Hotel. Fifty-six outstanding speakers will participate in the scientific program and panel discussions will be held on each day of the meeting.

Additional information may be secured by communicating with the Secretary, Dr. B. T. Beasley, 701 Hurt Building, Atlanta 3, Georgia.

WARNING

Information has been received that a man by the name of Roland, purporting to represent the American Medical Association, has been contacting nursing homes in Texas and Louisiana and threatening them unless they purchase his books.

Dr. George F. Lull, Secretary Manager of the American Medical Association has advised that no one by this name is employed as a representative of the AMA and that the AMA does not have agents in the field selling books. Members of the State Society are therefore warned against making purchases from such a person.

CARDIOVASCULAR DISEASES IN WOMEN MAY NOT BE INFLUENCED BY OBESITY

The development of high blood pressure and coronary diseases in women may not be influenced by obesity as it is in men, a study of 1,000 persons by three New York heart specialists disclosed.

The reason for this possible difference between the sexes is not obvious, the doctors wrote in the December 26 Journal of the American Medical Association.

"It is apparent that hypertension is found more commonly in obese men than in those who are of average weight or who are underweight, but the exact relationship between hypertension and obesity remains obscure," the doctors pointed out. "There is no definite evidence that the obesity is causally related to hypertension. It may be that the factors resulting in hypertension also produce obesity, e.g., emotional influences, faulty metabolism or a hormone disturbance.

DRINKING AND IMPORTANT ACTIVITIES DON'T MIX

If you have something important to do, don't drink beforehand.

The greatest danger from the use of alcoholic beverages concerns the relationship of drinking to subsequent actions, a medical consultant wrote in the Journal of the American Medical Association, stating:

"For example, the after-dinner drinks may be perfectly harmless if no important activities are undertaken at such times, whereas the drink taken before driving a car, running machinery, or performing any task that demands accurate decisions or mental acuity might be dangerous. The drinker should observe the following rule. For every two drinks, he should wait three hours before undertaking important activities."

Contrary to popular opinion, the mixing of alcoholic drinks does not increase the intoxicating effects of alcohol, since these symptoms depend on the actual amount of alcohol consumed and other factors, he stated. However, this old wives' tale concerning the mixing of beverages may be of value, since promiscuous sampling is likely to lead to greater consumption of alcohol, just as a great variety of foods may lead to overeating, he said.

LIKE ATTRACTS LIKE

States with the highest number of doctors per capita have the largest number of young men and women who want to enter medical school, according to statistics compiled by the Association of American Medical Colleges and reported in the October issue of The Journal of MEDICAL EDUCATION. It is suggested that the common economic, educational and cultural factors which attract doctors also stimulate students to become doctors.

DIAGNOSING BONE TUMORS IN CHILDREN

There can be no denying the fact that malignant neoplasms of bone currently represent one of the most important causes of death in children.

This is a report made by Dr. John F. Holt, of the University of Michigan, Department of Roentgenology, in the current (November) issue of *Radiology*.

"For the year 1948," Dr. Holt points out, "malignant neoplasms caused more deaths in children from 5 to 14 years of age than any other disease."

When one considers that primary bone tumors, including those cases of leukemia with bone involvement, constitute approximately 25% of all malignant growth in this age group, and that they are almost invariably fatal, then their true importance becomes evident," Dr. Holt adds.

PRELIMINARY TESTS PROVE NEW ANTIEMETIC DRUG EFFECTIVE

Nausea and vomiting caused by a wide variety of clinical conditions and by use of numerous drugs have been suppressed or controlled through administration of a new drug, known only as compound 2601-A, it was reported in the October 3 Journal of the American Medical Association.

This new antiemetic drug, synthesized chlorinated phenothiazine, has been used on 70 patients "with gratifying results," even where other antiemetic drugs have failed, according to Drs. Gale G. Friend and James F. Cummins, Boston.

"Without doubt, compound 2601-A has a powerful selective effect against nausea and vomiting, and is effective whether given orally or intramuscularly," the doctors stated.

STERILITY OF SURGICAL LINEN HELPED BY 2 BACTERICIDALS

Impregnation of wet surgical linen with the bactericidal agents Roccal and hexachlorophene has proved an effective means of preventing transmission of bacteria to wounds during surgery, according to Dr. Harry D. Propst of the Guthrie Clinic and Robert Packer Hospital, Sayre, Pa.

Writing in the *American Journal of Surgery* (86:301, Sept. 1953), he describes laboratory and operating room studies demonstrating that bacteria pass through wet surgical cloth, thereby exposing the surgeon's hands, instruments and suture material to contamination. Linen became wet from the surgeon's or patient's perspiration; from irrigating solutions; and from sterile water used in cleansing instruments.

Testing the usefulness of Roccal, a quaternary ammonium compound supplied by Winthrop-Stearns Inc., Dr. Propst reports that 1500 cc. of 10 per cent Roccal were added to the last rinse during washing of surgical linen to give a 1:2500 dilution. Cost of Roccal for a complete day's laundry amounted to one dollar, he says.

Analysis of a piece of treated linen showed 0.44 mg Roccal per gram of linen, or 0.07 mg. per square inch. In vitro studies demonstrated that Roccal-treated cloth inhibited bacterial migration even when wet. The bacterial counts of both wet and dry materials decreased, with the percentage of culture showing no bacterial growth being 85 per cent when Roccal was used, and 18 and 60 per cent, respectively, for untreated wet and dry linens.

No cases of allergy due to skin sensitivity were reported by the hospital nursing staff following application of Roccal. Other properties of the compound are: activity from a pH of 3.0 ranging up to the alkaline level, low toxicity and stability to boiling and autoclaving. Tests showed, Dr. Propst observes, that hexachlorophene was also capable of inhibiting transmission of bacteria through wet linen.

He emphasizes that although the problem has been long neglected, the study demonstrates that incorporating either bactericidal agent into the final neutral rinse in laundering surgical linen will "decrease the possibilities of contamination from this source."

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WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

Mrs. George Feldner of New Orleans has been elected president of the Woman's Auxiliary to the Southern Medical Association. This is the second time that a New Orleans woman has received this honor, Mrs. Wiley R. Buffington having been the first to hold this high position. Mrs. Feldner is noted for her outstanding service in medical circles. She began her activities in 1929 only a few months after her marriage to Dr. Feldner. At that time she was appointed to the Orleans Parish Medical Society Auxiliary Board. From there she went on holding many chairmanships until the war years.

In 1941, when Dr. Feldner went into the service Mrs. Feldner went with him to the many camps at which he was stationed; and there she was active in Red Cross and Civil Defense activities. After their return to New Orleans she held several offices in the Woman's Auxiliary to the Louisiana Academy of General Practice and was elected president of that organization in 1950.

In 1952 she was elected president of the Touro Auxiliary. Mrs. Feldner is a prolific writer having written various short stories that have been published and she will well be remembered for her script on the "Louisiana Purchase" which was the basis for the most successful pageant that Orleans Parish Auxiliary presented last spring. Her current works include a skit which was presented at the December meeting of the Orleans Parish Auxiliary based on the "Night Before Christmas". Her talents include designing and she has designed the presidents' and past-presidents' pins for Southern, State, and General Practice Auxiliaries. Auxiliaries from all over the state proudly hail Mrs. George Feldner.

ORLEANS PARISH

Woman's Auxiliary to Orleans Parish Medical Society entertained at a Christmas Party recently at the Orleans Club. A playlet was presented based on the "Night Before Christmas". Drs. Daniel J. Murphy, Branch Aymond, and Edwin Guidry were in the cast. Others participating were Mmes. Branch Aymond, Lloyd Kuhn, J. Theodore Brierre, and children of the auxiliary members, Mary Carroll Guidry, Linda Golden, Adrienne Wild, Michael Murphy, Catherine Murphy, Chris Rougelot, Beverly Danton, Mary Gaylord Sanders and Antha Ann Sanders.

IBERIA PARISH

On November 5, the Woman's Auxiliary to Iberia Parish Medical society presented a style show. Proceeds for the show went to the auxiliary's "Loan Closet" project. The Loan Closet provides hospital supplies for the sick in homes, such as hospital beds, chairs, crutches, and similar articles. These are loaned to the Iberia parish families without charge, whether the person is needy or not. It was explained

that in many cases hospitalization at home for several weeks requires special equipment that might be used only once in a lifetime. The style show which was a great success and which will allow the auxiliary to buy much new equipment was under the chairmanship of Mrs. Edwin Landry. Other members of the committee were Mmes. T. P. Sparks, I. W. Gajan, Jr., Leon Slipakoff, Henry Dautrieve, K. S. Motty, Jr., and O. P. Brown

Another activity of the Iberia Auxiliary is their future nurses clubs which have been sponsored in the six high schools in the city. Radio programs featuring members of the auxiliary speaking on the various projects and aims of the auxiliary are held periodically.

THIRD DISTRICT

Woman's Auxiliary to the Third District Medical Society entertained at a Christmas Coffee which was held at the Windson Room of Metairie Country Club. Fifty members and guests were present to meet Mr. Robert Tallant and hear his latest book "Love and Mrs. Candy" reviewed by Mrs. Robert Crager. Hostesses for the event were Mmes. Shelley Gaines, John Tanner, O. L. Pollingue and Allan Goldman.

LAFAYETTE PARISH

The Lafayette Parish Medical Auxiliary held its Christmas party in the home of Mrs. J. Boring Montgomery as the only social of the year. Members brought contributions of gifts to be distributed by Mrs. Montgomery to needy children of Lafayette. This is a project which the members have undertaken each year. Mrs. K. J. Burdin presided at the coffee table.

BOOK REVIEWS

The Book of Health; by Randolph Lee Clark, Jr., and Russell W. Cumley, Editors, Houston, Texas, New York, Elsevier Press, 1953. Pp. 836.

In the last century a family medical book which purportedly explained to the housewife "all there was to know" about health and medicine and surgery and particularly obstetrics; and the steps to be taken in an emergency, or in the birth of a baby unattended by a doctor, was sold throughout the length and breadth of the United States to people in the cities and towns and on isolated farms and plantations. Many of the mothers used information obtained from these digests and raised their families sometimes in the absence of doctors. This necessity no longer exists, but a ready source for authentic knowledge about health and medicine and the human body has not been available for the millions of well educated, highly intelligent Americans, in book form since the early part of this century,—until *The Book of Health* was recently published.

This book accomplishes in an easily understandable way a description of the human body and its workings; the various diseases which may affect it, and various measures which are used to correct the deviations from normal health. Cooperative efforts of some 242 top flight physicians, dentists, and biologic scientists in carefully reviewing the chapters have eliminated errors and absurd or unauthenticated fancies. The book is factual and accurate and it is most interesting. Intended for intelligent laymen it is nevertheless an excellent book for doctors. Perhaps the most valuable part of it for the physician is a section on medical history which is well done and is profusely illustrated by historically pertinent pictures and portraits.

The book can be readily advised as a source of reference and study for both the profession and the educated layman.

HOWARD MAHORNER, M. D.

Basic Principles of Clinical Electrocardiography; by Hans H. Hecht, Springfield, Illinois, Charles C. Thomas, 1950. Pp. 88. Price $2.00.

One of the many, many books devoted to this subject, it is well presented and rather easily understood. Its merit, as well as its disadvantage, is its brevity.

I. L. ROBBINS, M. D.

Heart and Circulation: Diagnosis and Treatment; by Meyer Sclar, M. D., F. A. C. C., assisted by Jacob Melnick, M. D., New York, Froben Press, 1953. Pp. 357, illus. Price $7.50.

The stated aim of Dr. Sclar, Brooklyn cardiologist, has been to present the basic concepts of diagnosis and treatment in the field of cardiology and peripheral vascular disease, in order to simplify the approach of the general practitioner and medical student to cardiac patients. The short text treats important aspects of the field very briefly and in a very elementary fashion, relying in part on a telegraphic method of presentation. In making recommendations on controversial or unsettled subjects, the author has relied on his own experience, so that references to the literature are omitted.

It is doubtful that this book will find wide usefulness and it is not recommended.

W. D. LOVE, M. D.

Shock Syndrome; Edited by Roy Waldo Miner, Annals of the New York Academy of Sciences. Vol. 55, Art. 3. Pp. 345-542. Price $3.75.

This is an excellent summary of the recent work and thinking on shock and related problems as presented by the investigators who have been most active in the field. The various papers were given in a conference on shock syndrome held by the Section of Biology of The New York Academy of Sciences on November 30 and December 1, 1951. The discussions which followed the presentation of the papers are also given.

The monograph is divided into 3 parts. Part 1 deals with various aspects on the shock syndrome and includes discussions of the role of coronary circulation, metabolic changes, adrenal cortex, electrolytes, etc. Dr. Jacob Fine and his colleagues present their recent findings emphasizing the importance of bacteria in the development of shock and General Sam F. Seeley has a short but instructive chapter on intra-arterial transfusion. Dr. E. B. Reeve's paper on "The Quantities of Blood Lost by Grossly Injured Men Within a Few Hours of Wounding" is a particularly good analysis of blood volume and hematocrit estimations.

Part 2 is devoted to plasma and volume expanders and includes experimental and clinical data on the various substances that are being investigated, such as Dextran, P. V. P., and globin. While considerable new data is available since the presentation of these papers, they serve to indicate the nature and directions of the experimentation that is being conducted as well as some of the inherent difficulties.

Part 3 is an interesting paper by Huber on the cold sterilization by electron beam as a possible tool for the inactivation of the virus of homologous serum jaundice in plasma. The problem of sterilizing plasma has been a particularly troublesome one and Dr. Huber's suggestions and data are very promising.

Much has been written and will continue to be written on this ever-present problem. At the present time, many scientific disciplines are being called upon to collaborate in the search for solutions of the various aspects of the problems. To present any coherent or reasonably complete picture is difficult, but this monograph does serve to indicate the progress that is being made and the opinions and points of view that are developing. As such, it is a valuable contribution.

H. S. MAYERSON, Ph. D.

Physiology of Exercise; by Laurence E. Morehouse, Ph. D. and Augustus T. Miller, Jr., Ph. D., M. D. 2d ed. St. Louis, the C. V. Mosby Company, 1953. Pp. 355, illus. Price $4.75

This text has been revised to include the new information on the physiology of muscular exercise which has been made known since World War II. It, also, has combined several chapters of the first edition, shifted the order in which some of the subject matter had been presented previously and added a moderate amount of new material. A significant improvement is a greater attention to the fact that the students for which the text is intended have only an elementary knowledge of basic chemistry and physics.

In general, the second edition is an improvement over the first, but it is questionable that all the changes made are improvements.

JOHN K. HAMPTON, JR.

Atlas of Surgical Exposure of the Extremities; by Sam W. Banks & Harold Laughman, Philadelphia & London, W. B. Saunders Company, 1953. Pp. 391, illus. Price $15.00.

This Atlas represents the culmination of many years of careful preparation and fulfills a long felt need for an authoritative, comprehensive description of surgical exposure of the extremities. It is somewhat unique in its format and layout in that various operative exposures are discussed by regions and each step in the entire exposure is beautifully illustrated. The exposures, or indications for each exposure, are discussed and a concise description of the procedure included on pages opposite the illustration.

The use of proper names to designate various incisions has been purposely omitted.

There are few, if any surgeons, who will not benefit from this beautifully constructed and edited book. It is obviously a required reference for any one training in orthopedics or general surgery, and will serve as an authoritative reference for both the experienced surgeon and the occasional operator in operative procedures on the extremities. Both the authors and the publisher are to be congratulated on this exceptional, excellent work.

JACK WICKSTROM, M. D.

Gifford's Textbook of Ophthalmology; by Francis Heed Adler, M. D. 5th ed. Philadelphia, Pa., W. B. Saunders Company, 1953, pp. 488. Price $7.50.

Five editions and fifteen years from the date of first publication, Adler's Gifford has become the handbook of ophthalmology for general physicians and a standard text for undergraduate students. Its scope is tuned to meet the interests and needs of physicians whose dealings with ophthalmic problems are of a general nature. Lucid descriptions of ocular pathology and the ocular manifestations of systemic disease encountered in daily practice are briefly presented. Physiopathology, which the author is so well

qualified to discuss, subtly flavors clinical descriptions, and clarifies the rationale of many therapeutic measures. Equivocal theories and dogmatisms give way to concepts having general acceptance. The sections dealing with the retina, and ocular manifestations of systemic disease have been greatly enlarged to include particularly pertinent material on hypertension, sclerotic vascular disease, and diabetes. The general physician will find this especially useful in correlating eye ground findings with data representing the status of the vascular tree elsewhere in the body.

Newer medicinal agents, such as cortisone, are added to the therapeutic armamentarium. Although treatment of specific disease is dealt with in the sections devoted to that part of the eye involved, the final chapter provides a list of therapeutic agents most commonly used in ophthalmology. The method of preparation, and the indications for use of these agents are listed.

Where a hundred words may fail, the excellent photographs with which the volume is profusely illustrated leave no doubt in the viewer's mind as to the appearance of clinical conditions demonstrated. The illustrations are, for the most part, from the author's clinical collection. A number of photographs, paintings, and charts depicting special subjects were obtained from authorities on those subjects, or other publications. The figures and colored plates numbered three hundred and seven.

Except for very general outlines of minor surgical procedures, descriptions of ophthalmic surgical techniques have been replaced by discussion of clinical indications for surgery. In keeping with the plan to direct the contents of this book on a need-to-know basis to general physicians, details of major surgical procedures have been omitted. Erudition has given way to practicality. This is further demonstrated by the absence of all documentation except illustration acnowledgements.

This little book makes available in concise form a large amount of practical knowledge to an important segment of the profession. Every medical library, regardless of the branch of medicine around which it is built, should contain a copy.

GUSTAV C. BAHN, M. D.

PUBLICATIONS RECEIVED

Drapkin Books, Linden, N. J.: Salt and the Heart, by Edward T. Yorke, M.D.

Charles C. Thomas, Publisher, Springfield, Ill.: Resuscitation of the Newborn, by Joseph D. Russ, M.D.; Coal Tar and Cutaneous Carcinogenesis in Industry, by Frank C. Combes, M.D.; Pelvic Relaxations and Herniations, by James M. Wilson, M.D.; Retinal Circulation in Man and Animals, by I. C. Michaelson, Ph.D.